ON 'VOMITING SICKNESS' IN JAMAICA

(Report of the XXXth Expedition of the Liverpool School of Tropical Medicine)

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(Received for publication 25 June, 1913)

PLATES XXIX-XXXIII

In December, 1912, the Committee of the Liverpool School of Tropical Medicine resolved to send me to Jamaica in order to investigate the nature of the disease called 'vomiting sickness,' prevalent in that island during the winter months, and responsible for a considerable mortality, chiefly amongst native children. It was considered of particular importance, for obvious reasons, to determine whether a relationship exists between 'vomiting sickness' and yellow fever.

The Committee of the School received, through the Colonial Office, the assurance that the Government of Jamaica would do all in its power to further the objects of the expedition.

I left Southampton on December 18th, on the R.M.S.P. 'Thames,' and arrived in Kingston, Jamaica, on January 10th, 1913. The Government, through the Superintending Medical Officer, Dr. J. Errington Ker, at once made suitable arrangements in order to facilitate my task. It was agreed that I should establish my headquarters in Kingston, where I could carry on laboratory investigations, and at the same time be in easy communication with all parts of the island, always being ready to go out into the country at a moment's notice. Instructions were issued to all the District Medical Officers, requesting them to report by telegraph all cases of 'vomiting sickness' which might come to their notice, and to make arrangements for post-mortem examination if the cases ended fatally before my arrival. These instructions were faithfully carried out, and on January 13th I performed my first post-mortem examination at May Pen. The only drawback, which was some-

times seriously felt, was that of the very considerable distances which I had to travel in order to reach a case. Cases often occurred out in the bush; in many of these cases the authorities took the trouble of transporting the patient or body to the nearest hospital or mortuary; but this was not always possible, and sometimes it was necessary to travel first several hours by rail, and then several hours in a buggy, besides walking the last part of the way. Two inconveniences resulted: that it was impossible to see even approximately all the cases occurring and reported; and, secondly, that I usually arrived late, in fact, as a rule, after the patient had died, and in some cases even somewhat late for a post-mortem examination. The transport of material to Kingston, for pathological and bacteriological examination, was also sometimes delayed more than was desirable.

Under these conditions I was unable to see more than about half the number of cases reported during my stay in Kingston. One part of the island, the parish of Trelawny, I was particularly anxious to visit, as the majority of the cases diagnosed by Captain Potter as yellow fever had been observed there. I had, however, no opportunity of doing so; very few cases occurred this year in Trelawny, and these I could, for various reasons, not reach in time. Otherwise I saw cases, sometimes alive, but more often after death only, in nearly all the various localities where the disease occurred, and my material includes, as far as I can judge, all the divers types which, with any degree of reasonableness, can be included in discussions on 'vomiting sickness.' Altogether, I saw sixty-two cases (besides several which could at once be otherwise diagnosed), a fairly large number, considering that my whole stay in Jamaica lasted only ten weeks. In the meantime I carried on laboratory work in the Laboratory of the Public Hospital, Kingston; here I had the invaluable assistance of the Government Bacteriologist, Dr. H. H. Scott, who received the material which I sent in from the country, incubated and examined many of the cultures, and always helped me in every possible way. Without his kind assistance I should have been obliged either to refuse a large number of calls or to leave the larger part of the laboratory work undone.

The described conditions of work in Jamaica made it impossible to investigate all epidemiological, clinical, anatomical and micro-

biological details in each case as fully as might have been desirable. The histological work was only just begun in Jamaica, but was almost wholly carried out in Liverpool, and so far under better conditions, as no fresh cases of 'vomiting sickness' were continually claiming the attention of the worker. But various reasons made it desirable not to devote too much time to this part of the investigation. Other work was waiting at home and it was desired that I should as soon as possible proceed to Africa on important investigations. Under the circumstances the necessary examination of sections was done to establish an anatomical diagnosis in doubtful cases and confirm it in others, and illustrations were prepared in order to show the more important histological changes, but no attempt was made to describe the microscopical appearances in all details. This would have been a work of years, whilst at present only about two months could be devoted to it. Instead of delaying the publication of the report it was therefore thought advisable to publish it as soon as possible, especially because there seemed to be no probability that a closer study of histological details, though in itself extremely interesting, would throw any further light on the one question of paramount importance, the question of etiology. I must, however, reserve for myself the right of publishing later on more detailed descriptions of some of the cases which present certain features of more than ordinary interest, though, I believe, without any bearing upon the question of the nature of 'vomiting sickness.'

On March 22nd I sailed from Kingston on the R.M.S.P. 'Oruba,' and on April 15th I arrived at Southampton.

The report is divided into the following sections:—

I. HISTORICAL NOTES

According to Potter (1912) the term 'vomiting sickness' has apparently been in use in Jamaica for many years. My own impression was the same, but I could obtain no definite information. Being a disease which chiefly attacks native infants and children, it seems quite likely that it has not attracted the attention of medical men to any considerable degree, as infantile mortality has been and still is very high, especially among the natives. Native children are often ill, and die, without medical attendance, and in cases of 'vomiting sickness' it must be admitted that parents have one particular and often very good reason for not applying for medical aid, namely the extreme acuteness of the cases which, in combination with the long distances in many cases, makes it useless to send for a doctor as the child is usually dead before he arrives.

In the medical literature 'vomiting sickness' is apparently mentioned for the first time by Turton (1904), who gives a general description of the disease, summing up in the following way:—

'We have here a disease which occurs only in the cold months, in country districts, among the poorer classes and unhealthy surroundings; may attack more than one member of the family, either at the same time or at intervals of hours up to a day or two; usually attacking children irrespective of sex; presenting a longer or shorter period of *malaise*, followed by vomiting, convulsions, and death in a few hours; showing, *post mortem*, signs of gastro-intestinal irritation, and usually infection, with ascarides in large numbers; killing by respiratory failure; and having a very small death rate in cases where early treatment has been given.'

Turton is of opinion that the post-mortem appearances are due to 'venous congestion,' and has, in fact, constantly found hyperaemia of the following organs: cerebral and spinal meninges, lungs, liver, spleen, kidneys, and stomach. Some importance is apparently given to the presence of Ascarides, which were found in nearly every case in the small intestines, sometimes in enormous numbers and forming knots, causing distension of the bowel and irritation of its wall.

In earlier years the same disease may have figured in the Health Reports under other headings, especially under that of cerebro-

spinal meningitis; this question is discussed by Potter (1912, Appendix A). In 1903 the term 'vomiting sickness' appears as a synonym of gastro-enteritis (Annual Report of the Superintending Medical Officer), and in the following years the disease is referred to as 'vomiting sickness.'

In 1905 the Superintending Medical Officer, Dr. J. E. Ker, draws attention to the prevalence of 'vomiting sickness' and to the uncertainty which reigns with regard to its nature; the great interest and importance of the matter are pointed out, and the following points are stated as certain:—

'1. The disease appears at a certain fixed time of the year, November to March, at a time when the temperature varies greatly from day to night.

'2. It rarely appears in town; none of the cases reported have come from towns.

'3. The people attacked are chiefly, but not always, children.

'4. It appears so suddenly and runs its course so quickly that medical men never hear of two-thirds of the cases until after death has occurred.

'5. Frequency with which several members of a family are attacked.'

Ker states that the affection has been returned under various names on the death certificates, and quotes 'Cerebro Spinal Meningitis, Gastritis, Cerebro Spinal Fever, Vomiting and Fits, Black Vomit, Vomiting and Pain, etc.'

After this introduction Ker publishes a number of reports on 'vomiting sickness' by several District Medical Officers who were in possession of considerable experience with such cases.

Dr. Cooke, Mandeville, writes, 'There is no such thing as "Vomiting sickness." The vomiting is simply a symptom as it is of many other diseases. The disease is acute congestion of the lungs, occurring in children under 5 years of age, and only in the poor, ill-fed, and almost naked.' The description of cases given by Cooke appears to bear out his contention.

Dr. Thomson, Chapelton, describes an outbreak of 'vomiting sickness which occurred after a period of unusually cold weather. 'Cases occurred at the same time in widely separated portions of the district. The disease has been more virulent than in previous

years, more adults have been attacked than in previous epidemics, and the death rate has been enormous. So rapid is the course of the disease, that in many cases there has been no time to obtain assistance. Very few cases of the disease are seen, only those occurring in the neighbourhood of the village are reported, the others being heard of only after death has occurred, being reported as sudden death to the police.' Two typical examples of the history are given as follows:—'1. A young child of healthy appearance is put to bed. The parents are roused during the night by a cry and find the child foaming at the mouth, retching, unconscious and cold, with twitching of the limbs, which passes into actual convulsions. 2. A young adult, more often female, leaves home for market—is attacked on the way with retching, vomiting, and intense weakness, becomes unconscious, and is brought home a corpse. Post-mortem examination of these cases reveals no naked-eye appearances of disease. The mucous membrane of the stomach may or may not be congested from the vomiting. There is never any trace of an ulcer. No irritant poison has ever been detected in the cases submitted for analysis. There are never any signs of disease in the kidneys or meninges.'

Dr. Earle, May Pen, describes the disease as apparently 'a severe form of gastritis, with rapid collapse and shock,' and states that it is 'very fatal, especially amongst children.'

A report by Dr. Bell, dated 1900, deals with the disease as observed in the same district, May Pen. He gives detailed descriptions of both clinical symptoms and post-mortem findings. Malaise with epigastric pain is followed by mucous vomiting, and prostration. After an interval of apparent improvement the vomiting returns and collapse sets in. Death may occur after a more or less rapid course. In some cases children were found during night with convulsions, which soon ended in death, or vomiting set in suddenly, with collapse, and death ensued in a few hours. At the autopsies hyperaemia of meninges, brain and liver was observed; the spleen was enlarged, but only slightly congested; the kidneys were intensely hyperaemic, and the stomach showed ecchymoses in the mucosa. The mortality was only about 2 per cent. if treatment was instituted early; otherwise, treatment was of no avail. The term 'epidemic gastritis' is not considered

satisfactory, and no definite opinion is stated with regard to the nature of the disease.

Dr. Tillman, Vere, states that it was 'almost entirely a question of food and clothing, as out of over nine hundred cases treated, not one case occurred among the coolie-children of the district.'

Tillman diagnosed the first case which he observed as one of meningitis, but later on he arrived at the conclusion that the meningeal hyperaemia was a secondary phenomenon, due to lung stasis and stasis in the venous system; the gastro-intestinal hyperaemia and irritation are considered phenomena of primary importance. The cause of the whole disorder was thought to be atmospheric. He reports on 144 cases observed, with only two deaths. He describes general malaise and sudden severe pain in epigastrium with violent vomiting, anxious expression, weak, rapid pulse, and slow and shallow respiration. The temperature is said to be often normal, but in most cases rising to 102 or 103, generally preceded by a rigor. Convulsions and death follow, unless treatment is resorted to, in which case the convulsions may be represented only by contractions of the feet, and develop no further.

The anatomical lesions found by Tillman were 'erythematous appearance' of the gastric mucosa, hyperaemia of liver, spleen, kidneys, and meninges, and hyperaemia and oedema of lungs.

The paper by Dr. Turton, Stony Hill, which is mentioned above, is also included in this report.

Dr. Calder, Santa Cruz, states that he has seen no cases exactly corresponding to the type of 'vomiting sickness' as described by his colleagues, but reports two cases in which vomiting was a salient feature, and in which he has performed post-mortem examinations. One was a child, the other an adult; pancreatitis, jejunal hyperaemia and haemorrhage, and petechiae in the stomach were observed in both cases.

It is obvious that various diseases may have been referred to in these reports. One difference is particularly striking between the views expressed in the reports of Bell, Tillman, and Turton, and those held by the other District Medical Officers. According to the latter, 'vomiting sickness' is an extremely fatal disease, whilst the type described by the latter is fairly benign, the mortality being as low as 2 per cent., or even less. It might be supposed that only

the latter had had the opportunity of seeing cases in the early stages, whilst the Medical Officers of the former group had always been too late, because of longer distances or for other reasons. As no specific treatment is indicated, it appears, however, entirely unintelligible that the character of the disease should have been so completely transformed by the measures described. Two possibilities remain, either that some observers have overlooked mild forms of the disease, or that others have included cases of a different nature. Neither possibility can be dismissed, but the latter seems perhaps the more probable. Many of the recovering cases would evidently, in the usual course of events, have been diagnosed as acute gastritis or gastro-enteritis, especially those with febrile temperatures and without collapse.

Dr. Ker concludes his report with the recommendation that special and systematic investigation into the question of 'vomiting sickness' should be made at an early date.

So far, in 1905, the entity of 'vomiting sickness' has not become established, a fact which must be borne in mind when discussing the following reports.

The Annual Report (1906) contains brief notes about 'vomiting sickness,' from various districts; it appears that the disease had been less prevalent and perhaps less malignant than in previous years. Two post-mortem reports, by Dr. Earle, are published; in both cases there was hyperaemia of meninges, lungs, liver, spleen, and kidneys. Petechial spots were observed in the brain substance, and subepithelial ecchymoses in the stomach. The descriptions are almost identical in the two cases, which are considered typical examples of the so-called 'vomiting sickness.'

In the 1907 Report it is stated that 'vomiting sickness' has been more prevalent and more severe than usual. In Clarendon some 80 or 90 deaths were actually reported, but many more are believed to have occurred. In Trelawny both adults and children were attacked, with fatal results. The need of a thorough investigation is again pointed out.

In 1908 'vomiting sickness' is said to have been little prevalent.

In 1909 the disease is reported present in Falmouth and Chapelton, but has evidently not been very prevalent.

In the 1910 Report exceptionally severe outbreaks are reported, and the following interesting table is given:—

'Number of deaths recorded as having been caused by Vomiting, Vomiting and Fever, Vomiting and Convulsions, etc.

Parish	Month							Total
	Dec., 1908	Jan., 1909	Feb., 1909	Mar., 1909	Dec., 1909	Jan., 1910	Feb., 1910	
St. Andrew	6	1	1	—	3	8	16	35
St. Catherine ...	9	4	3	2	22	10	1	51
Trelawny	15	38	13	7	7	12	9	101
Westmoreland ...	2	—	1	3	3	5	—	14
Clarendon	42	14	14	7	91	43	19	230
St. Thomas	—	1	—	—	4	8	1	14
Hanover	3	7	8	1	2	1	3	25
St. James	4	20	15	1	10	3	4	57
Manchester	7	13	3	3	47	19	1	93
								620

In the same Report the following statement is made by Dr. Ker: 'That many of these cases may be, and undoubtedly are, due to ackee poisoning, possibly cassava poisoning, worms, Meningitis, Bronchitis, Gastritis, and other diseases, there is little doubt, but beyond these there remains an uenxplained residue which needs explanation.'

In this connection the frequency of helminthiasis in children is mentioned; one District Medical Officer found, on administration of vermifuges, that out of 72 apparently healthy children only two expulsed no worms, whilst two passed more than a hundred ascarides each.

Cases and deaths are reported, in 1910, from many districts—it is evident that more attention is now being paid to the disease. Dr. Tillman reports 1,200 cases, the mortality not being stated.

Tillman modifies his earlier statement about coolie children: they do not escape altogether, but practically so; the different susceptibility is explained by the coolie children being better cared for than the children of the natives.

In the 1911 Report Dr. Ker abstains from discussion of the subject, awaiting the result of Captain Potter's investigations carried out during the corresponding financial year. Outbreaks of 'vomiting sickness' are reported from Stony Hill, Claremont, (St. Ann), St. Ann's Bay, Duncans (severe), Falmouth, Montego Bay, Newport, Crofts Hill, and Chapelton; in the report from Ulster Spring it is stated to have been 'practically absent.'

In 1912 no special reference to 'vomiting sickness' is made in the Annual Report, but two cases are stated to have died in Public Hospitals, the locality not being given.

II. RECENT OBSERVATIONS

It is obvious, from the data given in Section I, that a great many factors have been submitted for discussion as probable causes of 'vomiting sickness.' The existence of a separate morbid entity has been denied by several observers, but as a rule each observer has given his own view with regard to its nature, and these views have differed considerably—from helminthiasis to bronchitis, and from ackee poisoning to meningitis. The prevalent idea, however, has probably been that which was pronounced by Ker, that a group of unexplained cases remained when those were separated out which could be classified as well-known diseases. During the last year or so the discussion has taken on a somewhat more definite character.

Potter (1912), in a Colonial Office Report, published the first systematic investigation into the nature of 'vomiting sickness,' making the startling announcement that the majority of the fatal cases were yellow fever. This had probably been suspected by many, outside Jamaica, considering that the name of black vomit is given as a synonym of 'vomiting sickness' in Ker's summary, and remembering the existence in other West Indian Islands of the so-called 'fièvre à vomissements noirs des enfants,' by several authorities regarded as a form of yellow fever.

Potter does not declare that 'vomiting sickness' is identical with yellow fever; he agrees with other observers that the former name has been applied to a great many different diseases, and his report contains abundant evidence that this is the case. A large number of cases are included which have evidently nothing whatever to do with the type under discussion. Numerous cases are malarial, and others are otherwise definitely diagnosed. One group of seven cases, described in Appendix D to Potter's Report, shows very clearly the abuse, even of a most dangerous nature, to which an ill-defined name may lend itself. Five fatal and two non-fatal cases occurred in one hut, the District Medical Officer at first diagnosing 'vomiting sickness,' but afterwards suspicion of poisoning arose. One of the bodies was exhumed and arsenic was found in the organs. In spite of this evidence the investigation was not pursued, and the jury found that the cause of death was 'vomiting sickness.'

Eliminating from the discussion all these cases in which some other definite diagnosis can be made, Potter is of opinion that the remaining cases show the clinical characters and post-mortem appearances of yellow fever.

Before considering the question of yellow fever, Potter discusses the explanations previously advanced, and arrives at the conclusion that they are all unsatisfactory. With regard to cerebro-spinal meningitis he regards it as impossible that a large number of fulminant cases should develop without the simultaneous occurrence of other cases running a more prolonged course; besides, he says, in cases which had recovered, 'sequelae, such as paralysis, blindness and deafness should be expected,' and in fatal cases 'the post-mortem examinations would have shown definite inflammatory changes in the meninges.' The hypothesis of ptomaine poisoning has not been supported by symptomatological, nor by circumstantial evidence. The question of ackee poisoning as a cause of death—in some cases—is left open. Cassava poisoning is considered out of the question, as in that case the presence of prussic acid, the poisonous agent, would probably have been noticed.

Potter observed personally 73 cases, clinically or post mortem (the numbers run only to 70, but two different cases are both numbered 43, and the two cases in the additional section of

Appendix B were also seen by Potter); in addition he has collected a considerable number of observations from official reports. In many of the cases, both personal and from other sources, a definite diagnosis of malaria or some other affection was made, but a considerable number remains in which Potter suspects yellow fever, and in seventeen cases he feels certain of this diagnosis. In these seventeen cases the character of the stomach contents and the lesions of the gastric mucosa are certainly often suggestive of yellow fever, but the changes observed in other organs are usually so incompletely observed that the reader cannot possibly appreciate their nature and significance. It is very remarkable, and by no means in accordance with common experience in yellow fever, that the kidneys are often stated to be normal, and the urine free from albumin. The same objection which Potter makes against the assumption of the cases being cerebro-spinal meningitis can, with equal right, be made against a diagnosis of yellow fever: one would certainly expect that other cases occurred at the same time with more typical symptoms and a more prolonged course. To the reader of the report it is a great inconvenience that detailed descriptions are very seldom given, and that the findings recorded are not fully considered in the light of yellow fever pathology and epidemiology.

Potter's conclusions were at once contradicted by the Governor of Jamaica, in a letter to the Colonial Office. In support of his letter the Governor publishes a report by Scott (1912, 1), containing observations on cases of 'vomiting sickness,' with demonstration of meningococci in the spinal fluid. Later on, Scott recorded further observations in two Official Reports (1912, 2 and 1913, 1), and soon afterwards published a more detailed paper on the same subject (1913, 2).

In this last paper Scott summarizes his results. His first observations are on cases of meningitis of an ordinary type in which he found meningococci. Subsequently, he observed some cases of a fulminant type, showing the characters of 'vomiting sickness,' but occurring at a time of the year when this disease was usually not prevalent. All cases—five—occurred in one family. Investigation for meningococci was made in four out of the five cases, and the organism was found each time. In addition, meningococci

were found in the rhino-pharynx of a boy in the same family where the cases had occurred. Scott now followed up this line of investigation, and soon succeeded in finding the same organism in a considerable number of cases. These were all rapidly developing, but not all fatal; a considerable number belonged, undoubtedly, to the type of 'vomiting sickness.' Scott's paper deals with 52 cases, of which only a comparatively small number are personal observations, except for the examination of the spinal fluid, which he performed in 33 cases, the fluid either being obtained by himself or sent in by the Medical Officer in charge of the case. Meningococci were found in 23 cases, and similar cocci which could not be satisfactorily identified, in a few instances. In two cases spirochaete-like bodies were observed in the spinal fluid; both cases were of a rapidly developing type, and in one, at least, were meningococci also found. The spirochaete-like bodies were only seen on direct examination, but did not develop in cultures. The author leaves the question of their nature and importance open.

No anatomical lesions of any importance are mentioned in Scott's paper, except in the case of the meninges, and even in this case very few details are given. Little attention has evidently been paid to the pathology of the disease, which may, to a large extent, be accounted for by the fact that so few cases are personal observations.

Scott prepared vaccines from some of his cultures from the earlier cases, and used them later on in several mild cases and in a number of contacts—apparently with success. He fully realizes that vaccine treatment can have but little chance of success in the hyperacute cases, and, whilst recognizing the possible value of serum treatment, at the same time points out that its application meets with so many practical difficulties that it is impracticable in the majority of cases.

Scott's work furnishes a very definite basis for discussion. Since the appearance of his paper no further investigations have been published on the subject, but MacDonald (1913) has discussed and criticized Potter's paper, pointing out that he had failed to take up several important lines of investigation, and that no conclusive evidence had been brought forward in support of the diagnoses of yellow fever.

III. PERSONAL OBSERVATIONS

During my stay in Jamaica I observed 62 cases which were presented to me as cases of 'vomiting sickness,' and in which no other definite diagnosis could be made at once. A closer examination, however, revealed considerable differences, which make it necessary to divide the total number of cases into several groups for separate discussion. A brief summary of all the cases is given in the accompanying Table I; it has not been possible to include all symptoms and anatomical lesions, but, as it stands, I believe it conveys a fairly correct, general impression of the character of disease with which we have to deal in 'vomiting sickness.' Detailed histories of some of the cases—as far as they were obtainable—will be given further below when the various groups are discussed.

In addition to these personally observed and 'suspicious-looking' cases, I received from various colleagues observations which have not been included in the table, but some of them will be referred to later on. Nor have I included personal observations in which the symptoms at once suggested some other diagnosis than that of 'vomiting sickness,' unless the further course of events again appeared to bring this diagnosis in the foreground.

A few explanatory remarks are necessary in order to define more clearly the too brief headings of some of the columns in the table, and some indication of the pathological and bacteriological technique employed is also due.

First a general remark: the sign + indicates that the corresponding symptom was present, the sign −, that it is stated to have been absent. No sign means that no definite statement has been made in my notes; in this case, however, the symptom has usually been absent.

With regard to the clinical symptoms, it must be remembered that the majority of the patients did not come under medical observation during life. We therefore had to rely in these cases on layman's evidence, from which it, as a rule, was possible to elicit the presence or absence of symptoms as vomiting, convulsions, and coma. We found also that the statements with regard to the characters of the vomits usually appeared reliable. The vomited

Note.—Table I giving a synopsis of the cases will be found opposite p. 468.

matter was described, in the vast majority of cases, as watery or mucous, and sometimes it was said to have been greenish. With regard to black vomit, it was only described twice, and in two instances has its presence been queried. In one of the latter cases it was suspected because black stomach contents were found at the post-mortem examination, but nothing definite could be ascertained with regard to the appearance of the vomited matter. In the other case the grandmother of the patient, when telling the story of the child's illness, which had upset her considerably, to begin with mentioned black vomit, but afterwards denied emphatically to have said so, stating that the vomits had been absolutely colourless.

The column 'emaciation' has been inserted on account of the often-expressed opinion that 'vomiting sickness' is a disease chiefly or exclusively of emaciated or poorly nourished children. This opinion is emphatically contradicted by my experience, as the answers show. The sign + in this column does not exactly mean extreme emaciation, but simply a poorly nourished condition.

The column 'clinical character' gives the provisional diagnosis arrived at by the physician in charge and by myself, either from personal observation or from the information obtained from the parents or friends of the patient. The data given in the preceding columns are not always sufficient to bear out the diagnosis, which fact is explained by the peculiar circumstances under which we worked, especially that information often had to be obtained indirectly and from a class of people who were in many cases unable to give a definite answer to a concrete question, though a general impression was conveyed by their statements. In view of the vagueness of our opinion thus formed, the term diagnosis is avoided in the heading of this column.

In the column 'race,' bl. means black, and col. coloured; no cases were seen in whites, but the dictum that 'vomiting sickness' only occurs in the negro population was not confirmed, since two cases were observed in coolie children; the one case in a chinaman was of a different nature.

M. in the columns 18 and 19 means malarial parasites; P. means *Paraplasma flavigenum*.

The sign + in columns 19 and 20 means *Diplococcus meningitidis;* * indicates a growth of diplococci which differed from the

type of meningococci. The occurrence of other organisms, as staphylococci and B. coli, in a few cases, was regarded as accidental, and has not been tabulated.

In column 21, + indicates that albumin was found in the urine; in nearly all these cases hyaline or granular casts were also observed.

Most anatomical lesions are tabulated in accordance with the observations made at the post-mortem examination, but have, as a rule, been checked by histological observation. The statements with regard to 'inflammation' in liver, pancreas, and kidney refer exclusively to micro-cellular infiltration as observed by histological observation, and, consequently, do not not always correspond to the microscopical diagnosis of nephritis, etc., in the post-mortem reports; it is well known that the term 'acute parenchymatous nephritis' does not necessarily imply that cellular infiltration has already taken place.

Several organs, as thymus, thyroid body, pituitary body, ovaries, and others, have been entirely omitted from the table, as they never presented any lesions which appeared to have any essential bearing upon the question of 'vomiting sickness'; they were, however, automatically examined at the autopsies.

The post-mortem notes were dictated by me during the examination and written down at once by the Medical Officer or by an assistant.

The routine technique for histological examination was the following: Small fragments of the organs were fixed, at the autopsy, in sublimate alcohol (2 parts of saturated watery mercury bichloride solution, and 1 part of absolute alcohol), and in Flemming's strong osmic acid solution. Afterwards they were embedded in paraffin. Sections of sublimate-alcohol specimens were stained with iron-haematein, or with Friedlaender's or Delafield's haematoxylin, and counterstained with Hansen's aceto-picro-fuchsin solution, or with eosin. Other sections from the sublimate-fixed fragments were stained with Giemsa's solution or with Pappenheim's 'Panchrom,' according to the technique previously recommended (Seidelin, 1911, 1 and 2). In the case of the Flemming technique sections were stained with safranin.

The organs usually preserved for histological observation were

liver, pancreas, kidney, spleen, lymph nodules, and myocardium. Stomach, duodenum, adrenals, brain, and spinal cord were frequently examined, and other organs occasionally, according to special indications.

Examination for blood parasites was made in films of peripheral blood and heart blood, and from organs as spleen, liver, kidneys, and surface of brain. Microscopical examination was also made of the centrifugalization-deposit of the spinal fluid.

Cultures were made from the fluid obtained by lumbar puncture, in vivo or post mortem, or from the lateral ventricles of the brain, at the autopsy, and in many cases from the heart blood. Ascites-agar (nasgar) was the medium employed, and in addition to this, in some cases haemoglobin-agar or nutrient agar. Cultures were considered negative if no colonies developed within a week.

A positive diagnosis of *Diplococcus meningitidis* was made, if small, pale, greyish, semi-transparent colonies developed, consisting of Gram-negative diplococci, which produced acid in glucose, maltose, and galactose media, or changed at least one of these, but produced no change in other sugars. Similar organisms, which differed from the typical meningococcus in one or several particulars, were frequently met with, and will be the object of special discussion.

IV. DISCUSSION OF PERSONAL OBSERVATIONS

In order to discuss the cases tabulated in the preceding section, it will be convenient to state the characteristics which would appear to justify a diagnosis of 'vomiting sickness,' with the exclusion of other diseases. This means the provisional recognition of 'vomiting sickness' as a morbid entity, but the possibility must be left open that this entity may fall to pieces afterwards if it is found on further investigation that the cases may be classified under other headings. As characteristic symptoms I would regard: sudden onset; rapid development, the disease running its course to recovery or death in less than twenty-four hours, as a rule, in children, and in a few days in adults; vomiting; convulsions; coma; absence of fever, or only low fever; collapse; in fatal cases, anatomical lesions pointing to an acute intoxication, or hyperacute generalized

infection; negative result of the investigation for well-known pathogenic organisms.

This provisional definition having been laid down, the cases fall naturally into various groups. These groups will first be briefly discussed, and particulars of typical cases afterwards be given as illustrations of each group.

In the first group, A, all cases are included which correspond more or less closely to the above definition. The cerebro-spinal fluid has been examined in all cases, microscopically and by cultural methods; the result was always negative, as far as the *Diplococcus meningitidis* was concerned, but in some cases other microbes were found, especially *Diplococcus crassus* and other meningococcus-resembling diplococci. The blood was examined as far as possible, and in the cases where such examination was made the result was negative with regard to both malarial parasites and *Paraplasma flavigenum;* in all other cases the diagnoses of malaria and yellow fever were considered out of question, being unsupported or not sufficiently supported by the clinical and (or) pathological evidence. This group will, of course, form the principal object for the following general discussion; in this place it shall only be pointed out that the most constant and most remarkable pathological changes were: fatty metamorphosis of liver, kidneys, and other organs; necrobiotic changes of epithelia in pancreas, kidneys, and liver; swelling and hyperaemia of lymph nodules; hyperaemia of many organs, including the meninges, and a tendency to haemorrhages; widespread oedema of the connective tissues. The following cases belong to this group: Nos. 9, 14, 15, 16, 34, 37, 40, 41, 42, 46, 49, 52, 56, 59, 60, and 61; altogether 16 cases. Several cases, provisionally classified under other headings, may also belong to this group; case 58, for instance, closely resembles the cases here included, and would have been classified accordingly but for the reason that a few malarial parasites were found in the heart blood, an occurrence which is very likely to be quite casual.

Notes from the cases in group A are given below, some in full as types, others in abstract.

In the first case recovery took place; this case is, I believe, typical of the mild form of 'vomiting sickness.'

Case 9. A.S., 3 years, brown, 4, Chancery Lane, seen with Dr. Dryden in the Public Hospital, Kingston. January 21st, 1913.

Admitted in a weak condition; started vomiting at about 8 a.m., two hours before admission. Vomited several times in hospital; the vomiting consisted, as before, of abundant quantities of watery, frothy material, and took place without any effort. The impression was most nearly that of an acute obstruction of the pylorus. The child was stimulated, and reacted slowly in the course of the day, without any specific treatment. He appeared well the following day, and was discharged after two days, still somewhat weak and pale, but this seemed habitual.

Cerebro-spinal fluid was taken on the day of admission, at 2 p.m. Result: Temperature normal. Pulse frequent, slightly irregular, soft. No abdominal tenderness; no diarrhoea. No meningeal symptoms.

There is a suspicion that the child had eaten 'physic nut' before taken ill, but nothing definite is known, and the clinical picture did not correspond to that described in cases of poisoning with *Jatropha curcas*.

The following three cases, in three brothers, were seen by Dr. Joslen, of Annotto Bay. The first patient was also examined by me, and in the two other cases I performed the post-mortem examinations with Dr. Joslen.

Case 14. A.N., 12 years, brown, born in Jamaica and has always lived there. He was seen by Dr. Joslen at Evandale, St. Mary, where he lived in a poor hut, with a leaking roof, in the bush, together with his father and two brothers. The brothers were taken ill at the same time as A.N. (cases 15 and 16); the father was unwell for about a day, complaining of fullness in the chest.

A.N. was taken ill, suddenly, on January 26th, 1913, at 10 p.m., with vomiting, accompanied by considerable retching, slight epigastric pain, and slight headache. He soon became weak and drowsy. Thus he was found on January 27th, at about 5 p.m., and next day he was taken to the Hospital in Annotto Bay, where he arrived at 4.20 p.m. There I saw him the same evening, with Dr. Joslen.

January 27. Temperature 98·4° F. Pulse 120, regular, weak. Respiration 16, regular.

January 28, afternoon: temperature 98·4° F.; pulse 120. Evening: temperature 99° F.; pulse 88, regular, soft. No muscular rigidity; Kernig's symptom absent. Slight abdominal pain, corresponding to descending colon, but no tenderness. No enlargement of liver or spleen. Heart dullness and heart sounds normal. No sign of lung affection. The patient has no lice and no ticks.

January 29. Temperature normal; pulse 84, slightly irregular. The patient has slept well, feels well, and is hungry.

The patient recovered.

Lumbar puncture was performed in the evening of January 28; about thirty small drops of clear fluid were taken into two nasgar tubes, which remained sterile.

Blood smears were taken at the same time: they contained no parasites.

Case 15. C.N., 9 years, brown, born in Jamaica, and has lived there always. Evandale, St. Mary. Brother to Cases 14 and 16. Good health until about 10 p.m., January 26th, 1913, when he suddenly started vomiting. He became weak and drowsy, and died at about 5—6 p.m., January 27th.

Symptoms as in Case 16.

Post-mortem examination at 8 p.m., January 28th, 1913. Body of poorly nourished child. No jaundice. No rigor.

Lumbar puncture gives about thirty drops of clear fluid. *Spinal membranes*, both dura and pia, hyperaemic. Spinal cord shows no microscopical lesions.

Brain preserved unopened; the external aspect is normal. Dura mater cerebralis normal, pia considerably hyperaemic.

Hypophysis cerebri of normal size and aspect.

Abdominal cavity contains no fluid. Peritoneum of normal aspect. Abdominal organs normally situated.

Spleen: 18 × 11 × 3 cms. Capsule smooth, transparent. Colour reddish grey. Consistence diminished, slightly friable, not soft.

Right *kidney:* 9 × 4½ × 2 cms. Capsule easily detached. Surface smooth, without hyperaemia and haemorrhages. Consistence normal. Cortical substance pale. Slight hyperaemia of pyramidal bases.

Left kidney as right.

Mucosa of renal pelves, ureters, and bladder normal.

Genital organs normal.

Liver: 30 × 14 × 7 cms. Left lobe very long, which accounts for the excessive length of the organ, which is not essentially enlarged. Capsule smooth, transparent. Colour greyish red, with some paler, but not distinctly yellowish patches. The tissue is more uniformly grey. Consistence normal.

Pancreas: 12 cms. long, slightly hyperaemic; consistence soft.

Suprarenal capsules of normal aspect.

Pleural cavities contain no fluid. There are extensive, but delicate, adhesions of right pleura and similar, but less developed, of the left. *Lungs* hyperaemic in postero-inferior portions, contain air throughout. No infiltrations, no oedema, no infarcts. Slight anthracosis.

Bronchial lymph nodules slightly enlarged, anthracotic, and some of them fibrous, but no definite tuberculous phenomena are met with in lymph nodules or in lungs.

Larynx, *trachea*, and *bronchi* normal, except that frothy fluid escapes from the bronchi.

Tongue slightly coated.

Tonsils enlarged and the right considerably hyperaemic.

Oesophagus normal.

Thyroid body normal.

Pericardium contains no fluid. No pericardial, nor endocardial haemorrhages. Heart-conus: 6 × 8 × 2½ cms. Left ventricle fairly well contracted. Endocard and all valves normal. Myocard pale, of slightly diminishing consistence, shows fine yellowish stripes.

The *stomach* contains a considerable amount of brownish fluid, apparently not haemorrhagic, but corresponding to intestinal contents. The mucosa shows considerable hyperaemia, and on the anterior surface, near to the lesser curvature, are observed several petechiae.

The *duodenum* shows hyperaemia, but no haemorrhages. At a distance of about 2 cms. below the pylorus an ulcer is observed, which is of oval shape, 8 mms. long and 6 mms. wide, and is situated on the posterior wall. The ulceration has penetrated the mucous and muscular coats, and the bottom is formed only by the retro-duodenal tissues. The borders of the ulcer are slightly infiltrated, but there is no overlapping of the edges. The *jejunum*, *ileum*, *colon* and *appendix* without pathological phenomena.

Bile ducts and *gall-bladder* free from stones and obstructions; the gall-bladder contains greenish bile.

Numerous ascarides are present, about 35 in the small intestine, and one in the stomach. In the coecum a few *Tricocephali* are present.

Anatomical diagnoses: Hyperaemia meningum. Hyperplasia lienis. Hypostasis pulmonum. Metamorphosis adiposa hepatis. Pancreatitis parenchymatosa acuta. Sequelae pleuritidis duplicis. Gastritis acuta cum petechiis. Ulcus duodeni. Helminthiasis intestinalis.

The histological examination of the organs was of no particular interest, as post-mortem changes were marked. It may be mentioned that sections of the *liver* showed intense fatty change, which was irregularly distributed throughout the lobules; the tissues were anaemic, where fatty change was most marked, in other places capillary hyperaemia was observed. The *pancreas* showed marked oedema of the stroma and partial necrobiosis of epithelia. The *myocard* showed fragmentation and oedema of stroma.

A short, Gram-negative bacillus developed in the nasgar-tubes, inoculated with spinal fluid; no cocci.

Case 16. C.N., 5½ years, brown, born and lived in Jamaica, Evandale, St. Mary. Brother to A.N. and C.N., Cases 14 and 15.

The patient was seen by Dr. Joslen, Annotto Bay, January 27th, 1913, and the post-mortem examination performed by me on January 28th.

Apparently good health until January 26th, 10 p.m., when he suddenly started vomiting, and went on vomiting until death. Drowsy, listless and weak. Died at 5—6 p.m., January 27th.

No desire for food, but thirst. No chilliness, rigor, convulsions. No eye symptoms. Skin natural, no eruption, no jaundice. No fever. No lung symptoms. No headache. Perhaps slight cardialgia; considerable retching. No delirium, irritability, paralysis, rigidity, nor retraction of neck.

Post-mortem examination at 8 p.m., January 28th, 1913.

Body poorly nourished. Rigor mortis is present. No lice are observed. There is no jaundice. Superficial ulcerations are observed on both lower extremities.

Lumbar puncture gives 15 drops of blood-stained fluid.

The spinal *meninges*, both pia and dura, are hyperaemic, and the spinal cord is also somewhat hyperaemic.

The cerebral dura is normal, but the pia is considerably hyperaemic, especially on the convex surface of the brain. There is no fibrinous exudation. The lateral ventricles are not distended. There is no hyperaemia, and no other pathological lesions of the brain substance.

The *hypophysis cerebri* and the *thyroid body* are normal.

The *larynx*, *trachea*, and *bronchi* are normal. No pleural fluid, nor adhesions.

The *lungs* show hyperaemia in their postero-inferior portions, but neither oedema, nor infiltrations, nor infarcts; there is slight anthracosis. The bronchial lymph nodules are slightly enlarged and anthracotic.

The *heart* conus is 5 × 6 × 3 cms. The left ventricle is well contracted. The pulmonary artery contains no thrombi. All the valves are normal, as is the whole of the endocardium, and the pericardium. The myocardium is pale, otherwise normal. The pericardium contains a few cubic centimetres of clear fluid.

The *inguino-crural lymph nodules* are slightly enlarged, the axillary not enlarged.

The abdominal cavity contains no fluid; the peritoneum is of normal aspect. The abdominal organs are normally situated, with the exception of the kidneys. The sigmoid flexure is considerably distended with faeces.

The *mesenteric lymph nodules* are slightly enlarged ; they show no haemorrhages, and no signs of tuberculosis.

The diaphragm reaches on the right side the 5th, on the left side the 4th rib.

The *spleen* is 11 × 7 × 2 cms. Capsule smooth, transparent ; colour dark greyish red ; no haemorrhages ; consistence soft ; follicles grey, somewhat prominent. A supernumerary spleen is found in the gastro-splenic ligament.

The *suprarenal capsules* are normal.

The *kidneys* are united at their inferior extremities in the shape of a horse-shoe, with two separate pelves on the anterior surface. The capsule is easily detached. The surface is smooth, without any vascular distension. The renal tissue is normal ; there is slight hyperaemia of pyramidal bases. The mucosae of the renal pelves, ureters and bladder are normal. The bladder contains a considerable quantity of pale urine. The genital organs are normal.

The *liver* is 18 × 12 × 5 cms. The capsule is smooth and transparent ; colour uniformly red ; substance reddish grey without haemorrhages, and with no yellow patches. Consistence normal.

The *pancreas* is 10 cms. long, hyperaemic, and of diminished consistence.

The *stomach* contains alimentary substance and some mucus, but no blood. There are no haemorrhages in the mucosa.

Oesophagus, normal. *Tonsils* without signs of acute inflammation. *Tongue* slightly coated.

The intestines present no pathological changes. A large number of ascarides are present, but no other parasites.

The *bile ducts* are patent ; the gall bladder contains a considerable amount of greenish bile ; mucosa normal.

Anatomical diagnoses : Hyperaemia meningum. Hypostasis pulmonum. Lymphadenitis bronchialis, mesenterialis et inguinalis. Splenitis acuta. Pancreatitis parenchymatosa acuta. Lien accessorius. Symphysis renum. Helminthiasis intestinalis.

As in the foregoing case, post-mortem changes interfered with the histological examination. Sections of an inguinal *lymph nodule* showed marked hyperaemia and some lymphocyte-infiltration of the capsule. Sections of the *spinal cord* showed hyperaemia, but no cellular infiltration of the pia, and, apparently, oedema of pia and cord.

Short, Gram-negative bacilli develop in the nasgar tubes, inoculated with the spinal fluid ; no cocci.

The following is an example of vomiting sickness in a child of East Indian race, though, I believe, born in Jamaica. In this case, the presence of jaundice aroused suspicion of yellow fever, but after examination of stomach and liver this diagnosis was entirely dismissed.

Case 34. R.S., 4 years, coolie child, Windsor Park, St. Catherine, seen, after death, with Dr. A. G. Curphey, at the Hospital, Spanish Town, on February 6, 1913.

The child was admitted this morning, at 10 a.m., in a dying condition. He was said to have been taken ill suddenly the previous day, February 5, at 3 p.m., with vomiting. The vomits consisted of frothy, watery fluid, and were repeated the following morning. The child was crying and restless, but no shrieks were noticed. He had been healthy until this illness. In the hospital clonic convulsions were observed with intervals of about five minutes. There was no strabismus ;

no rigidity of neck ; no Kernig's symptom. Temperature 101·4°. The pulse was rapid, about 140, weak, regular. Frothy fluid escaped from the mouth, no vomiting took place in the hospital. A yellow stool was obtained after enema. The child died at 11.15 a.m. (Notes by Dr. Curphey, as I arrived not until about 2.30 p.m.).

The mother reports that *another child* of hers died suddenly on February 1, after having vomited watery, frothy fluid.

One child alive and in good health.

Post-mortem examination, at 3 p.m., on February 6, about four hours after death. Well nourished body. Rigor present. Slight jaundice. Slight enlargement of lymphatic nodules in neck, axillae, and inguina.

Lumbar puncture gives clear fluid, of which eight drops are taken in nasgar tube, and two smears are made. Spinal cord not examined. *Dura mater cerebralis* normal, *pia* slightly hyperaemic in posterior part of convex surface. No distension of lateral ventricles. *Brain* substance macroscopically normal throughout.

Hypophysis cerebri and *thyroid body* of normal size and aspect.

The *pleural cavities* contain no fluid, and there are no pleural adhesions. The *lungs* show slight hyperaemia of postero-inferior portions, but are otherwise normal. The bronchial lymph nodules are slightly enlarged and anthracotic. The bronchi, trachea, and larynx are normal.

The *pericardium* contains no fluid, and shows no petechiae. The heart is of normal size. The pulmonary artery is free. The endocard and all the valves are normal, the myocard is pale, greyish, of normal consistence.

The *peritoneum* is of normal aspect, and contains no fluid. Abdominal organs normally situated.

The *tongue* is slightly whitish coated. The tonsils are not enlarged. The oesophagus is normal. The *stomach* shows normal conditions ; there is no hyperaemia of the mucosa, and no haemorrhages. The *duodenum* shows considerable, and the *jejunum* slight swelling of lymphatic follicles ; no other abnormal conditions of intestinal tract. The bile ducts are patent ; the gall bladder contains a small quantity of greenish bile.

Liver : 20 × 11 × 5 cms. Capsule smooth, transparent ; surface reddish, with grey and yellowish patches ; the consistence is normal in the red portions, but diminished in the yellowish ones. The structure of the organ is homogeneous, without distension of vessels.

The *pancreas* shows slight hyperaemia, but is of normal size and consistence.

The *spleen* measures 6 × 4 × 1½ cms. Its capsule is smooth and transparent, its colour dark greyish red, and its consistence normal. The follicles are grey and prominent.

There is a slight hyperaemia of the left *suprarenal capsule*, but not of the right one, and both organs are otherwise normal.

The left *kidney* is 7 × 4 × 2 cms. The capsule is easily detached, the surface is smooth, dark, and shows capillary, but not venous, hyperaemia. The renal substance is dark red, without haemorrhages, and without yellow patches. The right kidney shows conditions similar to those of the left, except that the colour is slightly more pale.

The mucosae of renal pelves, ureters, and bladder are normal. The bladder contains about 50 c.c. of clear, yellow urine.

The *genital organs* are of normal aspect.

The *urine* is pale, clear, acid, contains a considerable amount of albumen, and a few casts, besides epithelial cells ; it contains no sugar, no bile pigment, no erythrocytes, and no leucocytes.

Anatomical diagnoses: Hyperaemia piae matris cerebri l.g. Hypostasis pulmonum l.g. Duodenitis follicularis. Jejunitis follicularis l.g. Lymphoadenitis mesenterialis, bronchialis, cervicalis, axillaris et inguinalis simplex. Metamorphosis adiposa hepatis. Hyperaemia pancreatis, glandulae suprarenalis sinistrae, et renum. Icterus.

Histological examinations:—

Liver: Marked capillary hyperaemia, slight microcellular infiltration of periportal connective tissue. No necrobiosis, and only very slight vacuolization of cells.

Pancreas: Marked venous and capillary hyperaemia, slight oedema of stroma, small patches of necrobiotic cells.

Spleen: Diffuse hyperaemia.

Kidney: Hyperaemia, which is very irregularly distributed, and often very intense; in a few places some interstitial leucocyte-infiltration; slight vacuolization of basal portions of cells in some of the convoluted tubules; in the same kind of tubules necrobiotic changes are common, with granular appearance of the protoplasm and more or less advanced karyolysis.

Duodenum: Enormous hyperplasia of Brunner's glands which not only increase the thickness of the submucosa, but also occupy large parts of the mucosa, even dislocating and compressing the Lieberkühn's crypts, and in some places reaching the free surface. There is also considerable hyperplasia of the lymphoid follicles and diffuse lymphoid infiltration of the mucosa.

Axillary lymph nodule: Diffuse hyperaemia and slight oedema of stroma.

Brain: Slight hyperaemia, no microcellular infiltration.

Spinal fluid: Very few cells, chiefly erythrocytes; some mononuclears, and a few polymorphonuclears.

Microbiological examination:—

Heart-blood: No growth.

Spinal fluid: Growth of a diplococcus, which forms large groups. It decolourizes with some difficulty by Gram's and Claudius's methods. It produces acid in glucose, maltose, galactose, lactose, and saccharose media.

Spleen: Growth of diplococcus identical with the one in the spinal fluid.

No parasites are found in smears from heart blood, liver, spleen, and spinal fluid.

In the following case there was no vomiting, but otherwise it appears to belong to the type of 'vomiting sickness.' There were no signs of meningitis or yellow fever, clinically or anatomically. No growth was obtained from the spinal fluid, and none in nasgar tubes from heart blood, but on haemoglobin-agar inoculated with heart blood there was a growth of diplococci, otherwise similar to meningococci, but showing the striking difference, that the colonies, both on haemoglobin-agar, and in subcultures or nasgar, were of a saturated whitish appearance.

Case 40. A.M.L.Y., 2½ years, black, Barbican, near Cherry Garden Gate, seen on February 12, 1913, with Dr. Croswell.

The child has enjoyed good health until this morning. She awoke at 6 a.m., apparently well, asking for water. Soon after, however, she complained of pain

in her stomach, and was seized by convulsions, apparently a kind of slow clonic contractions. She has suffered from no previous disease; in particular, she has had no cough, no fever, and no symptoms of worms. She was seen by Dr. Croswell at 8 a.m.; she was then comatose, and there was complete muscular relaxation. There was no abdominal retraction, no rigidity, and no Kernig's symptom. Temperature 97·8° F. No jaundice. The action of the heart was fairly strong. Injections of strychnin, digitalis, and nitroglycerin were given.

The child was in a dying condition, when I arrived at 10.45 a.m. The respiration was superficial, the pulse weak, but regular; it became immediately filiform, and could not be counted, but was not very frequent. There were no convulsions, and no rigidity, especially not of the neck, and no Kernig's symptom. The arms were faintly contracted in a semi-flexed position, but immediately afterwards became relaxed. The teeth were closed. There was grinding of the teeth. The child gave a faint shriek, and died at 10.50 a.m. At the moment of death, urine was passed in considerable quantity. The abdomen was considerably distended and this distension was said to have developed during the last ten minutes. There was no enlargement of liver or spleen.

About an hour after death spinal fluid and heart blood were taken in nasgar and haemoglobin-agar tubes, and smears were made.

Post-mortem examination (with Dr. Edwards, at 11 a.m., on February 13, about twenty-four hours after death). Well nourished, healthy looking body.

Dura mater cerebralis, and, particularly, spinalis considerably hyperaemic; pia mater slightly hyperaemic, mostly pronounced on convex surface of the brain and on the lumbar portion of the cord. No inflammatory exudation, and no free fluid in the lateral ventricles. The brain and spinal cord are of normal appearance.

The *inguinal*, *axillary*, and *cervical lymph nodules* are slightly enlarged, and intensely hyperaemic.

The abdominal cavity contains no fluid. The peritoneum is of normal aspect. The abdominal organs are normally situated. The intestines are considerably distended. The diaphragm reaches the fourth intercostal space on both sides.

The *spleen* measures 7 × 5 × 2 cms. The capsule is smooth and transparent, the colour dark red and grey. The follicles are grey and very prominent, the consistence normal.

Suprarenal capsules normal.

Right *kidney:* 6 × 4 × 1½ cms. Capsule easily detached; surface smooth, with venous and capillary hyperaemia; cortex hyperaemic, with yellowish grey patches, in its central portions; pyramids pale. Left kidney shows the same conditions as the right.

Renal pelves, *ureters*, and *bladder* of normal appearance. The bladder contains a small quantity of pale urine.

Genital organs normal.

Mesenteric lymph nodules considerably enlarged and slightly hyperaemic.

Liver: 23 × 13 × 4 cms. Capsule smooth, transparent; colour dark purple; substance uniformly reddish grey; consistence normal.

The *pancreas* is hyperaemic and of soft consistence.

The *stomach* contains a considerable quantity of bile-stained mucus. The mucosa is hyperaemic, but without haemorrhages.

The *duodenum* is hyperaemic. The jejunum shows a few hyperaemic patches and slight swelling of both solitary follicles and Peyer's patches. The swelling of the patches increases considerably in the ileum, especially in its terminal portion and is here accompanied by hyperaemia. The colon shows throughout its whole

length follicular swelling and on the top of each swollen follicle a black, minute spot. There are no parasites in the intestinal tract, except one small ascaris in the ileum ; in particular no parasites are seen corresponding to the swollen follicles in the colon. The bile ducts, gall bladder, and pancreatic ducts are of normal aspect. The gall bladder contains a small quantity of greenish bile.

The *oesophagus* is normal.

The *tonsils* are considerably swollen and hyperaemic, with haemorrhages in the tissue.

The *tongue* is covered, on the whole surface, with whitish fur.

The *larynx* is slightly hyperaemic, the trachea and the larger bronchi more pronouncedly so, and in the smaller bronchi, the mucous membrane is both swollen and hyperaemic, whilst there is an abundant mucous secretion. There is a pronounced hyperaemia of the postero-interior portions of both *lungs*, whilst the anterior portions are markedly pale.

The *bronchial lymph nodules* are slightly enlarged and hyperaemic, as well as the mediastinal. There are no signs of tuberculosis in either lungs or lymph nodules.

The *pleurae* are normal.

The *pericard*, *endocard*, and *valves* are normal. The *myocardium* is very pale.

The *hypophysis cerebri* and the thyroid body are normal.

The *thymus* measures $5 \times 4 \times \frac{1}{2}$ cm. ; it is of normal appearance and consistence.

Anatomical diagnoses : Lymphadenitis acuta. Tonsillitis haemorrhagica. Enteritis follicularis. Colitis follicularis et pigmentosa. Lymphadenitis mesenterialis m.g. Hyperaemia ventriculi, renum et meningum. Pancreatitis acuta parenchymatosa. Splenitis acuta follicularis. Hypostasis pulmonum. Bronchitis capillaris. Bronchitis, tracheitis et laryngitis incipiens. Anaemia myocardii. Helminthiasis intestinalis (*one* ascaris).

Urine : Contains small quantity of albumen and some hyaline casts.

Histological examination :—

Axillary and *cervical lymphatic nodules :* intense hyperaemia, venous and capillary. Moderate oedema of stroma. *Mesenteric lymphatic nodules :* oedema of stroma, but no hyperaemia.

Spleen : Intense diffuse hyperaemia.

Liver : Intense diffuse hyperaemia, moderate fatty changes equally distributed throughout the lobules, slight necrobiotic changes.

Microbiological examination :—

Nasgar tubes inoculated with *spinal fluid* and *heart blood* remain sterile, except one in which one colony of short Gram-negative bacilli develops.

Haemoglobin agar tubes inoculated with spinal fluid remain sterile, but not those inoculated with *heart blood*, in which saturated white colonies develop, consisting of Gram-negative cocci, mostly diplococci which grow abundantly, also in nasgar, and which ferment glucose, maltose, and galactose, but not lactose and saccharose.

The heart blood contains microscopically no organisms ; smears from liver, kidney, spleen and mesenteric lymph nodules contain various types of bacteria and those from liver and kidney contain also small diplococci. Smears from bone marrow contain no organisms.

Case 41. M.A.B., 3 years, black, Whitehall, with Dr. C. R. Edwards, Half-way Tree.

Seen alive by Dr. Edwards, not by me. Previous health good. Taken ill in the morning of February 13, 1913, with vomiting and convulsions; died at 11 a.m. on the same day.

Post-mortem examination, at 11 a.m., on February 14. Body fairly well nourished. No cutaneous affections, nor parasites. Rigor present. No jaundice. On the skin of the back a number of irregular, black patches, as from burns, and on the left hip a bulla, 5 × 2 cms.

Cervical, *axillary*, and *inguinal lymph-nodules* swollen and intensely hyperaemic.

Spinal fluid taken after death by Dr. Edwards, clear; only a small quantity was obtained. On opening the spinal canal the fluid was found somewhat thickened, slightly gelatinous. Cerebral dura shows slight, and spinal dura moderate hyperaemia. *Spinal* and *cerebral pia* shows considerable hyperaemia, mostly pronounced on the convex surface of the brain. No distension of the lateral ventricles. Cerebral and medullar substance normal.

Hypophysis cerebri normal.

Diaphragm on the right side at fourth, and on the left at fifth intercostal space.

Pleurae without fluid and adhesions. The *Lungs* are slightly hyperaemic, chiefly in postero-inferior portions. They contain several small, haemorrhagic infarcts, and numerous petechiae are seen on the pleura of the right inferior lobe. There is slight oedema of the inferior lobes of both lungs.

The *bronchial lymph nodules* are slightly enlarged and slightly hyperaemic.

The smaller bronchi show hyperaemia and swelling of their mucosa and considerable mucous secretion; similar conditions obtain in the larger bronchi, trachea, and larynx.

Thyroid body normal.

Pericardium, *pulmonary artery*, and *aorta* normal. Epicardium hyperaemic. Left ventricle well contracted, forms apex. Heart conus 5 × 6 × 2 cms. Endocard and valves normal. Myocard pale, of slightly diminished consistence.

Peritoneum normal, contains no fluid, abdominal organs normally situated. Appendix occupies a posterior, lateral, ascending position.

Mesenteric lymph nodules considerably enlarged and slightly hyperaemic.

Spleen: 7 × 5 × 2 cms. Capsule normal; colour dark purple, with grey spots; follicles slightly prominent, grey; consistence normal.

Suprarenal capsules normal.

Right *kidney:* 8 × 4 × 2 cms. Capsule easily deatched. Surface dark, with capillary hyperaemia; cortex hyperaemic, with yellowish grey patches in central portions. Pyramids pale. Left kidney shows the same characters.

Mucosa of renal pelves and ureters hyperaemic. The bladder contains a considerable quantity of pale yellow urine, of which some part is collected; the mucosa is normal.

The *oesophagus* is hyperaemic. The *stomach* contains a small quantity of a alimentary fluid; the mucosa shows slight hyperaemia. The *duodenum* shows considerable hyperaemia and some petechiae. The *jejunum* shows numerous hyperaemic patches and a few ecchymoses in its upper portions. *Ileum* pale, with considerable swelling, but no hyperaemia of Peyer's patches. *Appendix* 10 cms. long, normal. The *large bowel* shows a few hyperaemic patches, is otherwise normal. The jejunum, ileum, and colon contain an enormous number of guava seeds; also a few in appendix. One Tricocephalus in coecum. No other parasites.

Bile ducts and gall bladder are patent and of normal aspect.

The *liver* measures 22 × 11 × 4 cms. Capsule normal; colour purple grey, with a few yellowish patches; consistence and structure normal.

The *pancreas* is hyperaemic and of dimished consistence.

The *tongue* is covered on its anterior portion with a whitish fur, on the posterior part of its surface with a dark and slightly haemorrhagic coating.

The *tonsils* are enlarged, hyperaemic, and show in their tissue haemorrhages and purulent infiltration.

The *thymus* measures 5 × 3 × ½ cms., and shows normal aspect.

The *urine* is pale, clear, contains a small quantity of albumin and a few granular casts; no sugar, no bile pigments.

Anatomical diagnoses: Poly-lymphadenitis acuta. Sequelae combustionis. Hyperaemia meningum. Hyperaemia et infarcti haemorrhagici pulmonum. Petechiae pleurae dextrae. Laryngitis, tracheitis, bronchitis et bronchitis capillaris catarrhalis. Nephritis parenchymatosa acuta. Hyperaemia ventriculi et intestinorum. Ecchymoses duodeni et jejuni.

Histological examination:—

Liver: Intense hyperaemia of a diffuse character; marked fatty change; slight necrobiosis and slight microcellular infiltration of stroma.

Pancreas: Almost complete necrobiosis of cells, the nuclei not staining with haematein, and the cell limits badly defined.

Kidney: Intense necrobiosis of epithelia of convoluted tubules, the protoplasm of which is granular and the nuclei poorly stained or entirely unstained. Glomeruli and straight tubules fairly well preserved.

Myocardium: No changes observed in sections.

Lymph nodule: Diffuse hyperaemia; patches of necrobiosis.

Spinal cord: Pia mater shows oedema and patches of microcellular infiltration.

Tonsil: Localized necrosis and haemorrhages.

Microbiological examination:—

Large Gram-positive bacteria develop in cultures on nasgar from the spinal fluid; similar bacteria are seen in smears from the spleen, and in sections from various organs. No diplococci, in smears, or cultures. Culture-tubes (nasgar and haemoglobin-agar) inoculated with heart blood remain sterile.

In the following case, the clinical symptoms suggested a diagnosis of meningitis; this diagnosis, however, was not confirmed anatomically or microbiologically; the case may provisionally be referred to as one of 'meningismus.'

Case 46. E.B., 2½ years, black, Rodney Street, Admiral Town (near Kingston), seen at Public Hospital, Kingston, with Dr. Thomson.

Sudden onset of convulsions at about 7 a.m., February 17, 1913. No vomiting. Admitted to hospital at 9.45 a.m.; temperature 98° F.; patient unconscious; pupils dilated. Limbs rigid; head retracted, marked opisthotonus. Kernig's sign present, most marked on the right side.

Lumbar puncture made (Dr. T.): Water-clear fluid escapes under considerable pressure.

When examined later on, by Dr. T. and myself, at 12.30 p.m. the limbs are absolutely limp, and there is only a faint indication of Kernig's sign on the right side, whilst it is entirely absent on the left. Still later, it is absent on both sides. Pulse fairly strong, but very irregular, about 100.

Several convulsions occurred in the hospital, consisting chiefly in tonic spasms, and the child died at 4.30 p.m.

Post-mortem examination, at 11.30 a.m., on February 18, nineteen hours after death. Body well nourished. No skin affections, nor parasites. No rigor. No jaundice.

Cervical, axillary, and *inguinal lymph nodules* slightly enlarged and hyperaemic.

The *subdural space* contains a considerable amount of serous fluid at the basis of the cranium, and fluid is seen escaping from the spinal canal. Slight hyperaemia of cerebral dura ; spinal dura normal. Pia slightly hyperaemic, mostly so on the convex surface of the brain, and on the lumbar portion of the cord. No fibrinous exudation. The lateral ventricles are not dilated, and contain only a few drops of clear fluid. Brain and cord substance normal.

Hypophysis cerebri somewhat hyperaemic, not enlarged.

Pleurae normal. Both *lungs* contain a few small haemorrhagic infarcts, are otherwise perfectly normal, without hypostasis, and without anthracosis. Bronchi, small as well as larger, trachea, and larynx, normal. Bronchial lymph nodules moderately enlarged and hyperaemic, not anthracotic.

Thymus : 5 × 2 × 0·3 cms., of normal appearance.

Thyroid body normal.

Pulmonary artery and *aorta* normal. *Pericardium* contains a few c.c. of clear fluid. Left ventricle well contracted, forms apex. Heart conus $4\frac{1}{2} \times 5\frac{1}{2} \times 2$ cms. Epicard, endocard, and valves normal. Myocard slightly pale, and of slightly diminished consistence.

Diaphragm at fourth intercostal space on right, and at fifth on left side.

Peritoneum normal, without fluid. Abdominal organs normally situated.

Spleen : 10 × 5 × 2 cms. Capsule normal ; colour pale, with some dark patches, and a number of petechiae on the surface. Follicles prominent, light grey. Consistence normal.

Suprarenal capsules slightly hyperaemic, otherwise normal.

Right *kidney :* 6 × 4 × $1\frac{1}{2}$ cms. Capsule easily detached ; surface smooth, dark, shows capillary hyperaemia ; cortex grey ; pyramids slightly hyperaemic at bases, otherwise pale ; consistence normal. Left kidney : 6 × 4 × $2\frac{1}{2}$ cms., otherwise as the right. Renal pelves, ureters, and bladder normal ; bladder contains a small quantity of clear urine.

Genital organs normal.

Liver : 20 × 12 × 5 cms. Capsule normal ; colour of surface dark purple, with some yellow patches ; substance grey, likewise with yellow patches ; consistence normal.

Pancreas slightly enlarged, intensely hyperaemic, and of somewhat diminished consistence.

Tongue normal. *Tonsils* slightly enlarged, not hyperaemic. *Oesophagus* normal.

Stomach contains alimentary fluid ; mucosa normal.

Duodenum shows slight hyperaemia and swelling of follicles.

Jejunum normal. The lower part of the *ileum* shows considerable swelling of both solitary follicles and Peyer's patches ; the latter are hyperaemic. Appendix 7 cms., normal. Colon normal. In the lower part of the ileum two ascarides ; no other parasites.

Gall bladder and bile ducts normal ; the former organ contains greenish bile ; the latter are patent.

Mesenteric lymph nodules considerably enlarged, not hyperaemic.

Anatomical diagnoses : Poly-lymphadenitis, l.g. Hyperaemia meningum. Infarcti haemorrhagici pulmonum. Hyperaemia renum. Pancreatitis parenchymatosa acuta. Metamorphosis adiposa hepatis. Petechiae lienis.

Histological examination :—

Lymph nodules : slight hyperaemia. Moderate oedema of stroma.

Spleen : Marked oedema of stroma.

Liver : Intense fatty changes, moderate necrobiosis, both phenomena equally distributed throughout the lobules. The cells, when their limits are recognizable, are seen to contain a considerable number of large vacuoles (fat), but are in no case filled to distension with a single fat drop, nor do they contain large numbers of minute droplets. No hyperaemia. No inflammatory phenomena. Moderate oedema of stroma.

Kidney : Patches of intense hyperaemia ; epithelia of convoluted tubules in many places granular, and nuclei poorly staining or even absent, though the tubules are fairly well preserved. Glomeruli of normal appearances. Slight oedema of stroma. In osmic acid-preparations small fat droplets are seen in the epithelia of the convoluted tubules.

Brain : No hyperaemia.

In films made from the *spinal fluid* without centrifugalization no cells are seen. After centrifugalization very few cells which are mostly lymphocytes, also several polymorphonuclear leucocytes.

Microbiological examination :—

Nasgar tubes inoculated with spinal fluid ; one tube remains sterile and in another tube there is growth of (*a*) Gram-positive staphylococci, (*b*) Gram-negative diplococci forming chains, and changing maltose, but not glucose, galactose, saccharose and mannite. Heart blood : Nasgar tubes remain sterile ; on haemoglobin agar Gram-negative cocci develop similar to those in cultures of spinal fluid and showing marked formation of chains, but less marked diplococcal arrangement. No parasites are found on microscopical examination of spinal fluid, peripheral blood, heart blood, spleen and bone-marrow.

Case 49. M.A.W., 3 years 2 months, black, seen after death, with Dr. Earle, May Pen, who had seen the child on the previous day, when it was ill. February 19, 1913.

The child was quite well on February 17, and went to bed in the evening as usual, and slept well. On the 18th about 7 a.m. she was taken ill with convulsions which started suddenly ; there were contractions of arms and legs and rolling of the eyes ; no stiffness of neck observed. The attacks of convulsions followed each other rapidly. She was unconscious. She was seen by Dr. Earle at about 10 a.m. She was afterwards conscious and quiet until about 3 p.m., when convulsions again set in—about five occurred during the afternoon—and she again became unconscious and remained so until death, which took place at 9.45 p.m. She vomited about five times during the day, each vomiting occurring when a convulsion was about to set in.

Post-mortem examination, at 1 p.m., about fifteen hours after death. Body of well-nourished, absolutely healthy-looking child. No rigor. No jaundice. No cutaneous affection, and no parasites.

Spinal fluid flows freely by lumbar puncture, about 5 c.c. taken for examination ; it is perfectly clear.

The spinal venous plexus are extremely gorged ; dura and pia spinalis are moderately hyperaemic, pia especially in its lumbar portion ; no fibrinous exudation. Cord substance normal.

Dura mater cerebralis normal ; pia moderately hyperaemic, especially on posterior portion of the convex surface. No exudation. Lateral ventricles contain a few c.c. of clear fluid. Brain substance normal.

Cervical, axillary and *inguinal lymph nodules* moderately enlarged and slightly hyperaemic.

Diaphragm at fourth intercostal space on both sides.

Pleurae normal. *Lungs* contain a few small, haemorrhagic infarcts, and are slightly hyperaemic in postero-inferior portions; otherwise normal. The smaller as well as the larger bronchi, trachea, and larynx, contain a small amount of frothy mucus; otherwise perfectly normal.

The *pericardium* contains a small amount of clear fluid; heart conus $5 \times 6 \times 3$ cms. Pulmonary artery and aorta normal. Epicard, endocard, and valves normal. Myocard slightly pale, of somewhat pasty consistence.

Thymus: $9 \times 5 \times \frac{1}{2}$ cms. Appearance normal.

Thyroid body normal.

Peritoneum normal, without fluid. Position of abdominal organs normal; appendix inwards and downwards into the pelvis. Mesenteric lymph nodules moderately enlarged, not hyperaemic.

Spleen: $10 \times 1 \times 1\frac{1}{2}$ cms. Capsule normal; colour pale, with dark spots; follicles grey, slightly prominent; consistence normal.

Suprarenal capsules hyperaemic, of normal size.

Left *kidney:* $9 \times 4 \times 2$ cms. Capsule easily detached; surface pale, with somewhat dilated stellate veins; cortex grey with yellowish patches; pyramids slightly hyperaemic at their basis, otherwise normal. Right kidney, $8 \times 4 \times 1\frac{1}{2}$ cms., similar to the right one, but slightly more hyperaemic, and with more prominent yellowish patches.

Renal pelves hyperaemic, ureters and bladder normal; the bladder contains about 30 c.c. of clear urine.

Genital organs: hyperaemic, otherwise normal.

Liver: $20 \times 12 \times 5$ cms. Capsule normal; colour of surface distinctly yellowish with a few hyperaemic patches; tissue uniformly yellowish grey; structure normal; blood filling about normal; consistence somewhat diminished, pasty.

Pancreas: 11 cms. long; not hyperaemic; consistence considerably diminished.

The *stomach* contains a small quantity of alimentary mucus; the mucosa shows a few small patches of capillary hyperaemia.

Duodenum normal, as well as jejunum and ileum.

Colon shows a large number of black dots, corresponding to the centres of the follicles, which are not swollen.

The colon ascendens contains one Tricocephalus and two ankylostomes; no ankylostomes higher up in the intestine.

Bile ducts and *gall bladder* normal.

The whole surface of the *tongue* is covered with whitish fur.

Tonsils normal.

Oesophagus normal.

Anatomical diagnosis: Hyperaemia meningum. Poly-lymphadenitis. Hypostasis et infarcti haemorrhagici pulmonum. Metamorphosis adiposa myocardii et hepatis. Hyperaemia capsularum suprarenalium. Nephritis parenchymatosa acuta duplex l.g. Pancreatitis parenchymatosa acuta. Hyperaemia ventriculi l.g. Pigmentatio coli. Helminthiasis intestinalis.

Histological examination:—

Liver: Intense fatty metamorphosis, in some places most intense around portal vessels, in others equally distributed over all zones of the lobules. Capillary hyperaemia irregularly distributed. Slight necrobiotic changes.

Pancreas : No hyperaemia or cellular infiltration. No fatty change. The epithelia are as a rule normal, but in isolated places very poorly stained, the nuclei being almost invisible. Langerhans's islets well preserved and very definite.

Kidney : Some of the convoluted tubules show advanced granular and fatty metamorphosis, but in most the cells are fairly well preserved and the nuclei well stained. The glomeruli are considerably retracted.

Myocardium : slight fragmentation.

Spinal fluid : Numerous cells, mostly mononuclear ; only a few polymorphonuclear leucocytes.

Microbiological examination :—

Nasgar tubes inoculated with *spinal fluid* and *heart blood* remain sterile ; some sporulating Gram-positive bacteria develop in haemoglobin-agar tubes. In films from spinal fluid, after centrifugalisation, groups of diplococci are observed, also various bacteria.

Case 59. C.W., about 30 years, black, Barbican, seen after death, at Public Hospital Mortuary, with Dr. C. R. Edwards, of Half-Way Tree, on March 6, 1913.

Patient felt ill in the evening of March 5, vomited, had some whisky, felt better, woke up at about 1 a.m., on March 6, vomited again, had convulsions, became unconscious, and died about 6 a.m.

Post-mortem examination, on March 6, at about 2 p.m. Well-nourished, healthy-looking body. No skin diseases, nor parasites. No jaundice.

Lumbar puncture gives an abundant quantity of faintly cloudy fluid ; about 60 c.c. are collected, but the fluid is still flowing when the needle is withdrawn. Spinal dura, pia, and cord normal. Cerebral dura, pia, likewise normal, as well as the brain ; lateral ventricles not distended.

Hypophysis cerebri : 13 × 6 × 3 mms., slightly hyperaemic.

Subcutaneous fat intensely yellow, orange, not icteric, but reminding of the condition described from German Africa, as due to the ingestion of a certain kind of vegetable oil. Visceral fat of the same colour.

Cervical, axillary, and *inguinal lymph nodules* considerably enlarged and hyperaemic, the axillary mostly so.

Diaphragm at the fourth intercostal space on the right side, at the fifth on the left.

Both *pleurae* show total, fibrous adhesions. The *lungs* show slight hyperaemia and oedema of the postero-inferior portions. The right lung contains in its superior and inferior lobes several fibro-calcareous noduli. The smaller bronchi of both lungs show moderate hyperaemia and swelling of their mucosae, and some mucous secretion. Larger bronchi, trachea, and larynx normal.

Bronchial lymph nodules slightly enlarged and anthracotic.

Thyroid body normal.

Pericardium shows total, fibrous adhesions. Heart conus, 9 × 10 × 5 cms. Left ventricle fairly well contracted, forms apex. Pericardial fat shows several ecchymoses on anterior surface of right ventricle. The mitral valve shows several thickened, yellow patches on the anterior flap, and some nodular fibrous thickening of free edge. There is no stricture nor insufficience of the valve, and the other valves and endocard are normal. Myocard 1 cm. max. in left, 3 mm. in right ventricle ; it shows yellowish stripes and small patches ; consistence somewhat diminished. Pulmonary artery normal. The aorta shows extensive and intensely yellow patches of different sizes, some with superficial loss of substance, others with calcareous plaques ; the patches make their appearance immediately

over the aortic valve, are most prominent in the arch, and are continued far down into the abdominal portion.

The diaphragm shows over right lobe of liver a hard fibrous nodule, of about the size of a small walnut, and close to the nodule a small, serous cyst.

Peritoneum normal. Abdominal organs normally situated.

Spleen: 11 × 7 × 2 cms. Capsule adherent; tissue dark purple, with prominent grey follicles; substance soft.

Suprarenal capsules normal.

Left *kidney:* 11 × 6 × 3 cms. Capsule easily detached; surface smooth, pale grey, with patches of slight capillary hyperaemia; cortex pale grey, hyperaemic stripes and somewhat prominent glomeruli; columnae Bertini somewhat bulging forwards on the cut surface, which is pale yellowish grey; pyramids pale; consistence considerably diminished. The right kidney shows the same size and characteristics as the left one. Renal pelves, ureters, and bladder normal; the bladder contains about 500 c.c. of clear, pale urine.

Ovaries contain several serous cysts, no corpora lutea, and both ligg. lata contain also serous, multilocular cysts. Right tuba normal; left contains serous liquid in its considerably distended fimbriate extremity.

Corpus uteri: 9 cms; collum 5 cms., transverse diameter of corpus 8 cms. The perimetrium is normal, except for a small serous cyst on left surface. The endometrium cervicis appears normal, the endometrium corporis is slightly swollen and covered with muco-purulent secretion. The posterior wall of the corpus contains an almost globular mass, 6 cms. in diameter; consistence soft, elastic. Section shows a brain-like structure with some fatty masses. The anterior wall contains a small, submucous nodule, of similar aspect and consistence as the larger one.

Liver: 27 × 14 ×6 cms. Capsule normal; surface spotted, purple and dark yellow with smaller and larger hyperaemic patches, the whole aspect similar to that of nutmeg liver; consistence diminished, somewhat friable.

Bile ducts normal, patent; gall bladder normal, contains greenish bile.

Pancreas of normal size, not hyperaemic, of slightly diminished consistence.

Tongue coated with thick, white fur.

Tonsils normal.

Oesophagus normal.

Stomach contains alimentary fluid; the mucosa is hyperaemic in the neighbourhood of the lesser curvature, and in the cardiac portion, where several patches of petechiae are seen.

Duodenum slightly hyperaemic. Jejunum and ileum normal, except for a few patches of capillary hyperaemia. Appendix normal.

Colon shows patches of capillary hyperaemia.

No intestinal parasites.

Mesenteric lymph nodules slightly enlarged, not hyperaemic.

Urine: contains no albumen, no sugar and no casts.

Anatomical diagnoses: Pigmentatio telae adiposae. Poly-lymphadenitis acuta. Sequelae pleuritidis duplicis. Noduli calcareo-fibrosi (tuberculosi) pulmonis dextri. Bronchitis capillaris l.g. Sequelae pericarditidis. Endocarditis mitralis chronica fibrosa. Metamorphosis adiposa myocardii. Aortitis atheromatosa et calcarea. Nodulus fibrosus diaphragmatis. Splenitis acuta. Perisplenitis chronica. Nephritis acuta parenchymatosa duplex. Cystes ovarii utriusque. Hydro salpinx dextra. Cystis parva perimetrii. Tumor intramuralis uteri (Sarkoma) Tumor (metastaticus) endometrii. Metamorphosis adiposa et hyperaemia hepatis. Hyperaemia et petechiae mucosae ventriculi.

Histological examination :—

Lungs : Large fat drops are present in nearly all epithelial cells.

Pancreas : Most epithelial cells contain small fat granules. Extensive necrobiosis occurs, irregularly distributed. Langerhans's islets being affected as well as the epithelium proper ; there is considerable hyperaemia which is likewise irregularly distributed, in some cases apparently limited to Langerhans's islets ; there is also some infiltrating haemorrhage.

Kidney : Sections show considerable increase of connective tissue, especially in the pyramidal substance ; the arterial walls are fibrous and increased in thickness, as well as Bowman's capsules ; there are patches of hyperaemia and in some places interstitial haemorrhages. The epithelia of the convoluted tubules show in some places granular protoplasm, but contain no vacuoles. Their nuclei are in some places poorly stained or absent, but as a rule they are well preserved. In osmic acid specimens small fat granules are seen in nearly all epithelial cells in the convoluted tubules and also in the lining epithelia of glomeruli and Bowman's capsules.

Numerous ' mastzellen ' are seen in the interstitial tissues.

Uterine tumour : The tumour shows the structure of a fibro-myoma, but of somewhat atypical characters, being very rich in cells which show considerable polymorphism and often large ovoid or irregularly-shaped nuclei (sarcoma).

Microbiological examination :—

Cultures on nasgar from *spinal fluid* remain sterile for several days, later some Gram-positive staphylococci develop in one tube and diptheroid bacilli in another, Gram-positive staphylococci develop in cultures of *heart blood* (nasgar).

No parasites in smears from *heart blood.*

The following cases are similiar to the preceding ones and are therefore only given in abstract.

Case 37. G.B., 16 months, black, sister to E.B. case, Rock near Four Paths, with Dr. E. R. C. Earle, May Pen.

Sudden onset of vomiting at 10 a.m., on February 4, 1913. The patient is said to have had retraction of head, but no convulsions. Slight strabismus. Temperature normal ; pulse 140, small, slightly irregular. Death February 5 at 11 p.m.

Post-mortem examination :—

Spleen : Numerous subcapsular petechiae.

Kidneys : Pyramidal bases intensely hyperaemic. Consistence considerably diminished. The left kidney shows pronounced superficial hyperaemia and a few minute haemorrhages on the surface.

Liver : Surface pale, yellowish, and pronouncedly yellow in some parts ; in other places considerable hyperaemia. The consistence of the yellow portions is soft, whilst it is about normal in the hyperaemic patches.

The *stomach* contains a considerable amount of dark, grey, alimentary fluid, with no evidence of haemorrhage. Mucous membrane pale, of normal appearance. The duodenum shows several hyperaemic patches. The jejunum and ileum present likewise some hyperaemic patches, but the hyperaemia is much less pronounced than in the duodenal patches.

A few ankylostomes and a few ascarides are found in the upper portion of the jejunum ; no tricocephalus.

Anatomical diagnoses : Hyperaemia piae matris cerebralis et oedema incipiens. Lympho-adenitis universalis. Nephritis acuta duplex. Metamorphosis adiposa

hepatis m.g. Pancreatitis parenchymatosa acuta. Hypostasis pulmonum. Hyperaemia et ecchymoses pericardii. Metamorphosis adiposa myocardii. Helminthiasis intestinalis.

Microbiological examination :—

Spinal fluid : Nasgar tubes : Gram-negative diplococci, which decompose both glucose, maltose, galactose, lactose and saccharose after five days.

Case 42. C.W., 2½ years, black, Spanish Town Road, with Dr. C. R. Edwards, Half-way Tree.

The previously healthy child woke up at 4 a.m., on February 14, 1913, and vomited a small quantity of frothy mucus ; convulsions set in, and coma, from which she never recovered ; she died at about 8 a.m., at the D.M.O.'s (Dr. Edwards's) office, to which she had been taken just previously ; Dr. E. obtained blood smears, spinal fluid, and urine before death.

Post-mortem examination, at 3 p.m., on February 14, seven hours after death.

Spinal meninges and cord normal, not hyperaemic. Cerebral dura slightly hyperaemic, but pia of perfectly normal appearance. Brain normal.

Right *pleura* normal ; left pleura shows slight, but extensive adhesions and numerous petechiae. Both lungs show a few small haemorrhagic infarcts, but are otherwise normal, without hypostasis. Mucosae of smaller and larger bronchi, and of trachea, and larynx, normal.

Both *suprarenal capsules* slightly enlarged, and both, especially the left, show haemorrhagic infiltration.

Kidneys : Capsule easily detached ; surface smooth, with slight capillary hyperaemia ; cortex normal ; pyramids pale.

The *stomach* contains a small quantity of bile-stained mucus ; its mucosa is perfectly normal, as is also that of the duodenum.

The *liver* measures 23 × 14 × 5 cms. Capsule normal ; colour dark purple on the surface ; the tissue is grey, with slight increase of the portal connective tissue ; consistence on the whole slightly increased, but in some places friable.

Pancreas slightly hyperaemic, of normal consistence.

The *lymph nodules in the hepatic hilus* are very considerably enlarged, of greenish-grey colour, and of soft elastic consistence.

Urine contains no albumin, and no casts.

Anatomical diagnoses : Poly-lymphadenitis acuta. Hyperaemia durae matris cerebralis l.g. Sequelae pleuritidis sinistrae. Petechiae pleurae sinistrae. Infarcti haemorrhagici pulmonis utriusque. Splenitis chronica. Epinephritis haemorrhagica duplex. Enteritis follicularis l.g. Helminthiasis intestinalis (one *Ascaris lumbricoides*).

Histological examination :—

Liver : Advanced fatty change, practically all cells being filled to distension with moderately-sized fat drops. Some irregularly distributed capillary hyperaemia and very considerable microcellular infiltration, penetrating from the periportal tissues into the parenchyma.

Bacteriological examination :—

Spinal fluid : In nasgar tubes growth of Gram-negative diplococci which decompose glucose, maltose and galactose after two days, and lactose and saccharose after four days.

Case 52. J.E., 6 years, black, seen, after death, with Dr. C. R. Edwards, February 24, 1913.

The patient is a brother to Elisabeth E. (case 51), and was taken ill suddenly about 8 a.m., on February 22, shortly after his sister; in fact the family ascribed his illness to his having been frightened by seeing the sister's convulsions. He had several convulsions, but no vomiting. He died comatose, at about 2 a.m., on February 23. He was seen by Dr. Croswell.

Duration of illness: eighteen hours.

Post-mortem examination:—

Axillary and cervical nodules considerably, inguinal slightly, enlarged and hyperaemic.

The *stomach* contains alimentary fluid; mucosa normal, not hyperaemic.

Anatomical diagnoses: Hyperaemia meningum. Hyperaemia glandulae suprarenalis sin. et hypophysis cerebri. Hypostasis pulmonum. Splenitis acuta. Nephritis parenchymatosa acuta duplex l.g. Metamorphosis adiposa myocardii, hepatis, et pancreatis. Pigmentatio coli. Cadaverositas universalis.

The *histological examination* confirmed the anatomical diagnoses.

Bacteriological examination: No growth in nasgar tubes; in haemoglobin-agar tubes growth of Gram-negative bacteria.

Case 56. R.W., infant, black, seen with Dr. Gifford, at the Public Mortuary, Kingston, on March 3, 1913.

Post-mortem examination:—

Stomach shows considerable hyperaemia and a few petechiae.

Anatomical diagnoses: Hyperaemia piae matris cerebralis. Poly-lymphadenitis. Hypostasis pulmonum. Metamorphosis adiposa myocardii l.g., pancreatis, et hepatis l.g. Nephritis parenchymatosa acuta duplex incipiens. Gastritis ecchymotica l.g. Lymphadenitis mesenterialis. Helminthiasis intestinalis (*Ascaris lumbricoides*).

The *histological examination* confirmed the anatomical diagnoses except that the liver showed a rather intense fatty metamorphosis and some necrobiotic changes. The pancreas showed, in addition to fatty change, some localized hyperaemia, interstitial haemorrhages and fibrinous exudation; also slight oedema of stroma; the islets of Langerhans were of normal aspect.

Microbiological examination:—

No growth in nasgar tubes inoculated with *spinal fluid.*

Case 60. I.R., 4½ years, black, Four Roads, seen, after death, on March 7, 1913, at Kingston Hospital Mortuary, with Dr. C. R. Edwards.

Previous health good. On March 6, in the evening, 'she complained of abdominal pain, and later on she vomited. She went to sleep. Next morning she woke up early, screaming; she had convulsions, became comatose, and died.'

Post-mortem examination:—

Lumbar puncture gives clear fluid, with admixture of blood. Intra-spinal vessels intensely congested. Spinal dura and pia slightly hyperaemic. Cerebral dura normal, pia slightly hyperaemic, especially over cerebellum. Brain and cord normal.

Cervical, inguinal, and axillary lymph nodules enlarged and hyperaemic, especially the latter.

Stomach normal, contains alimentary fluid. Duodenum shows considerable follicular swelling, and the ileum shows slight swelling of solitary follicles and Peyer's patches in its lower part; the colon likewise shows follicular swelling; appendix 6 cms., normal.

Anatomical diagnoses : Hyperaemia meningum l.g. Poly-lymphadenitis acuta. Hypostasis pulmonum. Splenitis acuta. Nephritis acuta. Enteritis follicularis. Metamorphosis adiposa hepatis. Petechiae pericardii visceralis.

Histological examination of liver and kidneys confirmed the anatomical diagnoses.

Bacteriological examination :—

No growth in nasgar tubes (*spinal fluid* and *heart blood*).

Case 61. C.W., 12 years, black, Whitehall, seen, after death, with Dr. C. R. Edwards, at Kingston Public Hospital Mortuary, 8-III-i.

Was taken ill on March 6, with severe abdominal pain. No rise of temperature. No vomiting. No convulsions. Died March 7, at 4 a.m.

Anatomical diagnoses : Hyperaemia meningum. Hypostasis pulmonum. Poly lymphadenitis acuta. Hyperaemia et petechiae mucosae ventriculi. Helmin thiasis intestinalis. Cadaverositas universalis.

Bacteriological examination : Nasgar tubes remain sterile (spinal fluid).

Group B consists of cases in which 'meningococci' were found in the cerebro-spinal fluid, whilst definite inflammatory lesions of the meninges were absent, although a more or less marked hyperaemia was observed. Isolated cases of this nature, observed during an epidemic of cerebro-spinal meningitis, would be accepted under this diagnosis without much discussion. The peculiar circumstances in our case, and more especially the close resemblance of these cases to those of Group A, make it, however, necessary to consider very seriously the possibility that the diplococcus infection might be a secondary one. This question is one of the most essential of the whole problem and will be discussed together with the general aspects of 'vomiting sickness.' This group includes the Cases 1, 7, 8, 12, 20, 30, 33, 43, 44, 50, 55 and 57, a total of 12 cases.

As in the case of Group A, several cases are described in full, as types, and others only abstracted. Some of the cases are almost identical with those given as types in Group A. The post-mortem appearances of the meninges varied considerably in this group also, from slight hyperaemia to intense hyperaemia accompanied by oedema; in one case there was no hyperaemia at all and the meninges appeared perfectly normal. Similarly, the gastric mucosa showed in some cases normal conditions, in others hyperaemia, and in others again, both hyperaemia and petechiae. The conditions found in the stomach in Case 21 are particularly interesting.

Case 7. J.J.G., about 8 years, black, Chandler's Pen. Autopsy at 3.30-5 p.m., about twenty-six hours after death, January 18, 1913.

The child was healthy until the foregoing day—January 17—at about 4 a.m. when he was taken ill with vomiting, of watery fluid and food. He was conscious to begin with, but soon became unconscious, had twitching of the limbs, vomited a little again, and became gradually weaker, and died at 2 p.m. (he was not seen by medical man until after his death).

Post-mortem examination (with Dr. Earle). Fairly well-nourished child. Rigor present. Slight jaundice of sclerae.

Spinal puncture : about 20 c.c. of clear fluid.

Dura mater normal. *Pia mater cerebralis* shows slight diffuse hyperaemia.

Brain and *spinal cord* macroscopically normal.

Hypophysis cerebri appears likewise normal.

Tongue coated.

The *tonsils* are slightly enlarged, but show no evidence of acute inflammation.

Larynx, *trachea*, and *bronchi* contain bloody, frothy mucus, otherwise normal.

Bronchial lymph nodules enlarged and anthracotic.

No adhesions but a few c.c. of clear fluid in each of the pleural cavities.

The *lungs* show slight hyperaemia of the lower lobules, and some oedema of right superior lobe, but no infiltrations. No tubercular phenomena in lungs or lymph nodules.

Thyroid body normal.

About 10 c.c. of clear fluid in the pericardial cavity.

Heart : $5 \times 6 \times 2\frac{1}{2}$ cms. No pericardial haemorrhages. Myocardium slightly pale, shows no evidence of fatty metamorphosis.

No liquid in abdominal cavity. Peritoneum appears normal.

Spleen : $11 \times 6 \times 2\frac{1}{2}$ cms. Capsule smooth, transparent. Colour dark.

Suprarenal capsules normal.

Left *kidney*, $8 \times 4 \times 1\frac{1}{2}$ cms. Capsule easily detached. No superficial hyperaemia, but on section hyperaemia of pyramidal bases, which stand out very dark on the pale cortical substance. Right kidney as left.

Renal pelves and *ureters* normal.

The *bladder* presents a normal mucosa, contains a considerable amount of clear urine.

Liver : 21 × 11 × 5 cms. Capsule smooth ; transparent. No haemorrhages. Consistence normal, homogeneous. Lobuli very prominent, of an intense yellowish red colour.

The *pancreas* is somewhat enlarged, and is the seat of considerable hyperaemia. Substance extremely soft.

Oesophagus normal.

The *stomach* contains alimentary fluid. The mucosa is hyperaemic, especially in the vicinity of the lesser curvature. In this region numerous minute petechiae.

The *duodenum* shows very slight hyperaemia, but no haemorrhages. Jejunum, slight hyperaemia which becomes less pronounced lower down and is not at all observed in the ileum. No enlargement of solitary follicles nor of Peyer's patches. Four ascarides in the small intestine, and a few tricocephali in coecum. No ankylostomes.

Bile ducts and *gall-bladder* show normal mucosa ; the bladder contains dark brownish bile.

The large bowel and *vermiform appendix* show normal conditions.

Anatomical diagnoses : Hyperaemia piae matris cerebralis l.g. Tonsillitis chronica. Anthracosis lympho-bronchialis. Nephritis parenchymatosa subacuta.

Hyperaemia et petechiae mucosae ventriculi. Helminthiasis intestinalis (*Ascaris lumbricoides*, *Trichocephalus trichiurus*).

Histological examination :—

Mesenteric lymph nodules : oedema of stroma.

Liver : Marked fatty change, evenly distributed throughout the lobules. Hyaline and oedematous thickening of the stroma, corresponding both to portal and to central veins.

Pancreas : Interstitial oedema.

Spleen : Oedema of coarse trabeculae.

Kidney : No oedema, but slight increase of stroma.

Microbiological examination :—

Nasgar tubes inoculated with spinal fluid show growth of Gram-negative diplococci which change glucose and maltose but not saccharose, lactose, galactose and mannite.

Case 12. A.M., 3 years, black, at 'Four Roads,' St. Andrew (seen with Dr. L. O. Croswell).

The girl has been subject to colds in the head, but has otherwise been healthy. She has never passed any worms, according to her family. In the morning of January 21, 1913, she appeared slightly unwell, and at noon-time she started vomiting clear fluid and continued vomiting during the afternoon. She had no febrile symptoms, but at 5 p.m. convulsions set in. Dr. Croswell saw the patient at 5.30 p.m. ; she was then comatose and limp, showing general muscular relaxation. There was no rigidity, no Kernig's symptom, and no abdominal retraction. Four or five seizures were observed, lasting for about half a minute each time, and consisting in tonic, rather than clonic, contractions. There was seen froth at the mouth, and a sort of hiccough, but no vomiting. She died at 6.15 p.m., apparently from respiratory failure ; the pulse could be felt, and the heart went on beating for some time after breathing had stopped altogether.

The pulse was rather weak during the whole of the time during which the child was under observation. Adrenalin was given subcutaneously, as a stimulant, by Dr. Croswell.

Two years ago a brother of the deceased (then about 11 years old) had similar symptoms, but recovered under stimulating treatment. This boy was taken ill at a time when an epidemic outbreak of vomiting sickness reigned at 'Four Roads.'

Post-mortem examination, at 10 a.m., on January 22, with Dr. Edwards. Body well nourished. No rigor. No jaundice.

Spinal fluid is taken by lumbar puncture ; it appears clear, but is mixed with blood.

Tongue clean. *Tonsils* normal. *Larynx*, *trachea*, and *bronchi* without pathological changes, and empty, except the secondary bronchi which contain a small quantity of frothy mucus.

No adhesions, and no fluid in the pleural cavities.

Lungs slightly hyperaemic in postero-inferior parts. No oedema, and no infiltrations. No infarcts.

Bronchial lymph nodules not enlarged, of pinkish colour.

No anthracosis, and no tubercular phenomena in lungs and bronchial lymph nodules.

Heart : $3\frac{1}{2} \times 4\frac{1}{2} \times 2$ cms. About 5 c.c. of clear fluid in pericardium. Pulmonary artery free. Left ventricle well contracted. Myocardium pale,

greyish, without haemorrhages and yellowish patches. Consistence slightly diminished.

Thyroid body and *Thymus* macroscopically normal.

Peritoneum of normal aspect. Position of abdominal viscera normal.

Spleen : 6 × 3½ × 1 cms. Capsule smooth, transparent. Colour dark, with pale, prominent follicles. Consistence normal.

Mesenteric lymph nodules considerably enlarged, of pinkish colour, with slightly prominent vessels. Consistence normal.

Suprarenal bodies macroscopically normal.

Left *kidney :* 6 × 3½ × 1½ cms. Capsule easily detached. Surface smooth, stellate veins hardly visible. Cortex pale, especially in proximal parts. Small yellowish patches irregularly distributed in cortical substance. Pyramidal bases slightly hyperaemic, apices pale. No haemorrhages. Consistence slightly diminished. Right kidney : Same dimensions and aspect as left, only slightly paler.

Renal pelves, *ureters*, and *bladder* normal, the latter containing a few c.c. of pale urine.

Oesophagus normal.

The *stomach* contains a small quantity of alimentary fluid. The mucosa is slightly swollen and shows diffuse hyperaemia, mostly pronounced in the vicinity of the lesser curvature. In this portion of the mucosa there are a few groups of minute petechiae.

Duodenum : Slight hyperaemia.

Jejunum, *ileum*, and *large bowel* pale, without ulcers and haemorrhage. Solitary follicles enlarged in ileum, and jejunum, and Peyer's patches enlarged and hyperaemic.

Liver : 17 × 11 × 5 cms. Capsule smooth, transparent. Colour irregular, pinkish, with yellowish patches of different sizes, and a few hyperaemic patches. Substance homogeneously yellowish grey ; the small blood vessels hardly visible. Consistence slightly diminished, not friable.

Pancreas : 10 cms. long. Tissue slightly hyperaemic, and of considerably diminished consistence.

Bile ducts and *gall bladder :* Mucosa normal. Considerable amount of dark greenish yellow bile in bladder.

Genital organs normal, except that both ovaries contain small serous cysts.

Meninges : Dura mater normal. Pia mater spinalis normal ; pia cerebralis slightly hyperaemic, in particular on the convex surface. No oedema and no inflammatory exudations. Lateral ventricles not enlarged, contain a very small quantity of clear fluid.

Brain and *spinal cord* macroscopically normal.

Anatomical diagnoses : Hyperaemia piae matris cerebri. Hypostasis pulmonum levi gradu. Anaemia et metamorphosis parenchymatosa myocardii. Hyperaemia oedema levi gradu et petechiae minimae mucosae ventriculi. Enteritis follicularis. Anaemia et metamorphosis adiposa hepatis. Anaemia renum (et metamorphosis adiposa ?). Pancreatitis parenchymatosa acuta.

Histological examination :—

Liver : Intense fatty metamorphosis and very slightly marked necrobiotic phenomena. Marked increase of connective tissue, especially around portal vessels, but also penetrating into the lobules, separating small groups of cells from each other ; considerable microcellular infiltration, especially in the portal stroma.

Spleen : Intense diffuse hyperaemia ; follicles show necrobiotic changes.

Brain : No hyperaemia, no perivascular cell-infiltration.

Kidney : Marked granular appearance and moderate vacuolization (fat) of epithelia of convoluted tubules ; marked karyolysis in the same class of epithelia ; glomeruli considerably retracted. Marked increase of stroma, especially in pyramidal portions, slight microcellular infiltration.

Pancreas : Patches of beginning necrobiosis ; Langerhans' islets somewhat prominent.

Microbiological examination :—

Heart blood, nasgar tubes remain sterile for four days. Sanguinolent spinal fluid : Gram-negative diplococci (in the original culture the results of Gram and Claudius staining were somewhat irregular, in subculture the results were always negative), which change glucose, maltose, and galactose but not lactose, saccharose, and mannite. Spleen smears show small numbers of plump bacilli and a few chains of diplococci.

Case 20. T.J., 12 years, coolie (Hindoo), May River, St. Mary, seen on January 31, 1913, with Dr. Joslen, Annotto Bay. Was living in a dirty hut, with his father, mother and two brothers, all in a half-starved condition. The father was ill with stomatitis, vomiting and dysenteriform diarrhoea, and the mother was dying from phthisis, with acute pneumonia. The brothers were in good health. According to later information, the father recovered but the mother died.

Water supply from river ; no latrines ; house on the side of a steep hill, drainage consequently good.

Deceased retired to bed on the evening of January 30, apparently well. Next morning he was found dead in his bed. (Clinical notes by Dr. Joslen).

Post-mortem examination, on January 31, at about 6 p.m. Poorly-nourished body. Rigor present. No jaundice. No cutaneous affections, but numerous insect bites and scratches on lower extremities.

Spinal puncture gives no fluid. Dura normal. Slight diffuse hyperaemia of pia spinalis and cerebralis, mostly pronounced over cerebellum. No oedema, no exudation. Lateral ventricles not dilated. Brain and cord normal.

Pleurae normal. Lungs slightly anthracotic, otherwise normal.

Bronchial lymph nodules normal.

Larynx, *trachea*, and *bronchi* are hyperaemic and contain frothy fluid.

Thyroid body pale, otherwise normal.

Pericardium normal. Endocardium and valves normal ; myocardium likewise, as well as pulmonary artery and aorta.

Peritoneum normal. Abdominal organs normally situated.

Spleen : 16 × 10 × 3 cms. Capsule normal ; colour dark greyish red ; consistence semi-fluid. Tissue uniform ; follicles greyish, not prominent.

Suprarenal capsules normal.

Left *kidney :* 8 × 5 × 1½ cms. Capsule easily detached ; surface smooth, reddish with large yellowish patches ; stellate veins not distended ; tissue uniformly greyish red, with yellow patches in cortex. Consistence slightly diminished ; the right kidney presents the same characters as the left.

Liver : 20 × 15 × 6 cms. Capsule normal ; colour reddish grey, with yellow patches, and on the right lobe a number of minute haemorrhages ; consistence normal.

Pancreas hyperaemic, soft.

Tongue heavily coated. Tonsils without signs of acute inflammation.

The *stomach* shows on the central portion of the anterior wall eight dark, almost black, blood clots embedded in the mucous membrane ; when removed a red spot is left, but no actual opening of a vessel is to be seen. No hyperaemia of the mucosa and no petechiae.

Duodenum normal. The small intestine contains in many parts, especially in the middle part of the jejunum, numerous small, half-digested blood coagula, which are freely movable ; no haemorrhages in the mucosa, no ulcers, no follicular swelling and no hyperaemia. Appendix and large bowel normal.

Anatomical diagnoses : Hyperaemia piae matris. Splenitis acuta. Pancreatitis acuta parenchymatosa. Nephritis parenchymatosa acuta duplex l.g. Metamorphosis adiposa hepatis l.g. Haemorrhagiae ventriculi.

Histological examination :—

Spleen : Diffuse hyperaemia.

Liver : Localized hyperaemia. Cell infiltration of portal connective tissue. Moderate fatty change. No oedema of stroma.

Pancreas : Oedema of stroma. Necrobiotic changes of parenchyma.

Kidney : Slight oedema of stroma. Advanced necrobiotic changes of epithelia in convoluted tubules.

Microbiological examination :—

Spinal fluid, nasgar : Gram-negative diplococci which change glucose and maltose, but not lactose, galactose, saccharose, and mannite.

Case 44. E.P., 2 years, black, Kings Gate, seen, after death, with Dr. C. R. Edwards, Half-way Tree.

Previous health good. Sudden onset of vomiting, followed by convulsions, death in 3-4 hours.

Post-mortem examination, February 15, 1913. Body fairly well nourished ; rigor present ; no jaundice ; on the buttocks and lower extremities numerous scars as from scratching and small furunculi. Lice in hair, no other cutaneous parasites.

Lumbar puncture gives about 3 c.c. of clear fluid. Dura and pia spinalis show slight hyperaemia. Dura cerebralis normal ; pia shows slight hyperaemia of convex surface ; no exudation. Brain and cord normal. No tubercular affection.

Inguinal, axillary and *cervical lymph nodules* moderately swollen ; some of the inguinal and cervical nodules hyperaemic.

Diaphragm at fourth intercostal space on the right side, at the fifth on the left.

Pleurae without adhesions, contain no fluid ; the right pleura on the inferior lobe shows a number of miliary tubercles.

Lungs slightly hyperaemic, especially in the postero-inferior portions ; left lung otherwise normal. The right lung shows in its middle and inferior lobes numerous peribronchial tubercular nodules.

Bronchial lymph nodules on the right side are considerably enlarged and show caseous changes.

The smaller bronchi of the right lung show in many places caseous infiltration ; those of the left lung, as well as the larger bronchi, trachea and larynx show hyperaemic swelling of the mucosa and considerable mucous secretion.

Thymus : $9 \times 5 \times \frac{1}{2}$ cms ; appearance and consistence normal.

Thyroid body normal.

Pericardium contains a few drops of clear fluid. Pulmonary artery and aorta normal. Epicard, endocard and valves normal. Heart conus $5 \times 5 \times 2\frac{1}{2}$ cms. ; left ventricle fairly well contracted ; myocard slightly pale, of normal consistence.

Peritoneum normal, without fluid ; abdominal organs normally situated.

Tongue covered with white fur, on its whole surface. Tonsils slightly enlarged ; no signs of acute inflammation, no haemorrhages and no tubercular phenomena.

Oesophagus normal.

Stomach contains alimentary fluid ; mucosa shows follicular swelling, is otherwise normal.

Duodenum shows intense follicular swelling. Jejunum, ileum and appendix normal. Colon shows slight follicular swelling and punctate pigmentation corresponding to the folliculi.

The *bile ducts* are patent; their mucosa is normal, as well as that of the gall bladder, which contains a moderate quantity of greenish bile.

Liver: 19 × 12 × 5 cms. Capsule normal; colour dark purple; tissue shows a few yellowish patches, is otherwise normal.

Pancreas of normal size and colour, but of diminished consistence.

Spleen: 9 × 4 × 1½ cms. Capsule normal; colour normal; consistence soft; the tissue contains a number of miliary tubercles.

Suprarenal capsules normal.

Left *kidney:* 7 × 3 × 2½ cms. Capsule easily detached; surface smooth, pale, with considerable distension of stellate veins; cortex pale grey with yellowish patches; pyramids slightly darker, but not hyperaemic; consistence diminished. A few miliary tubercles in the tissue. Right kidney as the left, except that it does not contain any tubercles. Mucosa of renal pelves, ureters and bladder normal.

Genital organs normal.

Urine: faintly acid, contains a small quantity of albumen and a few granular casts.

Histological examination:—

Cervical medulla: Moderate hyperaemia.

Spleen: Diffuse hyperaemia; thickening of arterial walls. No histological tuberculosis.

Myocardium: Moderate fatty change.

Lungs: Caseous and miliary tuberculosis.

Bronchial lymph nodules: Hyperaemia; caseous and miliary tuberculosis.

Liver: Fatty change, in some parts moderate, in others intense.

Pancreas: Localized hyperaemia, partial necrobiosis, prominent Langerhans' islets.

Kidney: Diffuse hyperaemia, granulated protoplasma of epithelia in some convoluted tubules and nuclei sometimes poorly stained.

Stomach: The epithelial cells are as a rule fairly well preserved; some of them considerably swollen (hypersecretion ?). Glands are seen beneath the ordinary level of the mucosa completely embedded in connective tissue, but apparently not penetrating the muscularis mucosae which has been displaced, following as a continuous layer underneath the glands. Lymphoid tissue is scarce; there is moderate hyperaemia.

Case 50. J.M., 3 years, black, Old Hope Road, St. Andrew, seen at Kingston Public Hospital, with Dr. Thomson, February 21, 1913.

Admitted, at 12 noon, on February 21, with a history of convulsions of sudden onset, from early in the morning on the same day.

She is unconscious; there is no rigidity of the limbs; head not retracted; muscles of neck somewhat rigid; Kernig's sign not obtained. Knee-jerks absent. Pupils somewhat dilated, no reaction to light. A fresh, small burn is seen on the left wrist (hot bottles had been applied at home).

Temperature 98·4. Breathing laboured. Moist râles over front of chest. Heart's action very rapid and irregular. Abdomen not distended, nor retracted. Bowels act after enema. No vomiting.

The temperature went down to 98·2 later on; the child had several attacks of convulsions, during which the extremities were in a state of tonic contraction, and

were moved slowly to and fro ; no clonic movements were observed. One attack was observed for at least ten minutes, and lasted probably longer.

The child died at 5.30 p.m.

Post-mortem examination : February 22, at 10.30 a.m., seventeen hours after death. Body fairly well nourished. Slight rigor. No jaundice. No skin affection.

Cervical, axillary and inguinal lymph nodules considerably enlarged and somewhat hyperaemic.

Spinal fluid not obtained, as skull had been opened by assistant.

Dura cerebralis and *spinalis* normal ; pia moderately hyperaemic on convex surface of brain ; spinal pia not hyperaemic, but shows dark pigmentation. Cord and brain substance normal ; the lateral ventricles contain a few drops of serous fluid, from which culture is made.

Hypophysis cerebri normal.

Pleurae normal. Both lungs show slight diffuse hyperaemia, and, on the cut surface, mucous secretion escaping from the capillary bronchi. The smaller and larger bronchi, and trachea, and larynx are considerably hyperaemic, and contain mucous secretion.

Bronchial lymph nodules moderately enlarged and hyperaemic. No tubercular changes.

The *pericardium* contains a few c.c. of clear fluid. Heart conus $4 \times 5 \times 2\frac{1}{2}$ cms. ; left ventricle somewhat flabby ; epicard, endocard and valves normal. Myocard pale, with yellowish patches, of diminished consistence. Pulmonary artery and aorta normal.

Thymus : $9 \times 3 \times \frac{1}{2}$ cms. ; of normal appearance.

Thyroid body normal.

Peritoneum normal, without fluid. Abdominal organs normally situated. Appendix in internal position, between the small intestines.

Diaphragm at third intercostal space on right side, at fourth on left.

Mesenteric lymph nodules considerably enlarged, but not hyperaemic.

Spleen : $7 \times 4 \times 1$ cms. Capsule normal ; numerous subcapsular and intraparenchymatous ecchymoses ; tissue pale ; follicles not prominent ; consistence slightly diminished.

Left *suprarenal capsule* hyperaemic, of normal size ; right normal.

Left *kidney :* $7 \times 4\frac{1}{2} \times 2\frac{1}{2}$ cms. Capsule easily detached ; surface smooth ; pale, with patches of capillary hyperaemia ; cortex pale yellowish grey and columnae Bertini even more pronouncedly so ; pyramidal bases intensely hyperaemic. Consistence considerably diminished.

Renal pelves, ureters and *bladder* normal ; the bladder contains a small quantity of pale urine.

Tongue covered on the whole surface with thick, whitish fur.

Tonsils enlarged, without signs of acute inflammation.

Pharynx hyperaemic.

Oesophagus normal.

The *stomach* contains alimentary fluid ; mucosa normal, except a few hyperaemic patches.

Duodenum normal. Jejunum shows numerous hyperaemic patches ; no follicular swelling. Ileum shows moderate swelling of Peyer's patches, and slight swelling of solitary follicles. All parts of colon show a large number of superficial erosions ; colon descendens shows many hyperaemic patches.

Eight ascarides in jejuno-ileum ; no other parasites.

Liver : $21 \times 11 \times 4$ cms. Capsule normal ; surface dark purple, with large

yellowish patches ; tissue in some places dark, hyperaemic, but more often yellowish grey, and in some places distinctly yellow ; consistence diminished and somewhat friable.

Pancreas of normal size, intensely hyperaemic, and of diminished consistence.

Anatomical diagnoses : Hyperaemia meningum. Laryngo-tracheo-bronchitis catarrhalis. Bronchitis capillaris diffusa catarrhalis. Poly-lymphadenitis acuta. Metamorphosis adiposa myocardii et hepatis. Pancreatitis parenchymatosa acuta. Nephritis parenchymatosa acuta duplex. Hyperaemia capsulae suprarenalis sin., ventriculi et intestinorum. Haemorrhagiae lienis. Ileitis acuta. Helminthiasis intestinalis.

Histological examination :—

Liver : Intense fatty change, leucocyte groups in blood vessels.

Kidney : Moderate diffuse increase of stroma. Epithelia fairly well preserved except in a few convoluted tubules, where their protoplasm is granular and partially destroyed and the nuclei are undergoing karyolysis. The glomeruli are well preserved. Osmic acid preparations show small fat droplets in the basal portions of most of the epithelia of the convoluted tubules.

Stomach : Intense localized hyperaemia of mucosa.

Pancreas : Some parts of the sections show advanced necrobiotic changes of both the alveolar epithelia and the islets of Langerhans ; in other parts all cells are well preserved. The limit between well-preserved and necrobiotic cells is often quite sharp, cells of the two types being seen sometimes side by side in the same lobule or even in the same alveolus.

Microbiological examination :—

Nasgar tubes, fluid from lateral ventricles of the brain : Gram-negative diplococci which change glucose, galactose, and maltose, but not lactose and mannite. No growths in tubes inoculated with heart blood. No micro-organisms in smears of peripheral blood and heart blood.

Case 57. B.M., 5 years, black, seen, after death, with Dr. J. Huntly Peck, Spanish Town, March 5, 1913.

No clinical data obtainable.

Post-mortem examination, at 12 noon. Body fairly well nourished. No rigor. No jaundice. No skin affection, nor parasites.

Spinal puncture gives no fluid, even though repeated directly through the intertransversal ligaments in the lumbar and cervical regions.

Dura mater spinalis slightly hyperaemic, pia considerably hyperaemic, and dark-pigmented, especially on the sides. Cord normal.

Dura cerebralis normal, pia considerably hyperaemic, especially on the convex surface. Brain substance slightly hyperaemic.

Cervical, axillary and inguinal lymph nodules considerably enlarged and some of them hyperaemic.

Pleurae normal. Lungs slightly hyperaemic in postero-inferior parts. The smaller bronchi show slight hyperaemia of mucosa and mucous secretion ; larger bronchi, trachea and larynx normal.

Bronchial lymph nodules slightly enlarged and hyperaemic.

Thymus : $9 \times 4 \times \frac{1}{2}$ cms., of normal aspect.

Thyroid body normal.

The *pericardium* contains a few c.c. of clear fluid. Epicardium intensely hyperaemic, but shows no haemorrhages. Heart conus $5 \times 6 \times 2\frac{1}{2}$ cms. Left ventricle well contracted, forms apex. Myocardium slightly pale ; endocardium and valves normal. Pulmonary artery and aorta normal.

Diaphragm at fourth intercostal space on both sides.

Peritoneum normal. Abdominal organs normally situated.

Spleen : 9 × 6 × 2 cms. Capsule normal ; colour dark slaty ; consistence slightly diminished.

Suprarenal capsules normal.

Left *kidney :* 6½ × 4 × 2 cms. Capsule easily detached, surface smooth, pale, with slight distension of stellate veins ; cortex pale greyish ; pyramids normal, consistence normal. Right kidney, same characters.

Renal pelves, *ureters* and *bladder* normal ; the latter contains a considerable quantity of pale, clear urine.

Genital organs normal.

Mesenteric lymph nodules considerably enlarged and slightly hyperaemic.

Tongue coated, on its whole surface, with thick white fur.

Tonsils normal.

Oesophagus normal.

Stomach shows patches of capillary hyperaemia.

Duodenum normal. Lower down in the intestine hyperaemic patches.

Bile ducts and *gall bladder* normal, patent. Bile green.

Liver : 23 × 12 × 6 cms. Capsule normal ; colour uniform, greyish purple, tissue of normal aspect.

Pancreas hyperaemic, soft.

Hypophysis cerebri normal.

Anatomical diagnoses : Hyperaemia piae matris. Poly-lymphadenitis acuta. Bronchitis capillaris l.g. Pancreatitis parenchymatosa acuta. Hyperaemia renum l.g.

Histological examination :—

Axillary glands : Intense hyperaemia, oedema of stroma.

Spleen : Intense pigmentation.

Pancreas : Intense necrobiosis in patches, oedema of stroma.

Liver : Intense fatty change.

Kidney : Diffuse hyperaemia, more intense and with haemorrhages in some places ; beginning necrobiosis of epithelia in convoluted tubules. Oedema of stroma.

Microbiological examination :—

Cultures on nasgar (fluid from lateral ventricles of the brain) : Gram-negative diplococci, small groups, which change glucose, galactose, and maltose, but not saccharose, lactose, and mannite.

The following cases, of which only very brief notes are given, were similar to the above types of group B.

Case 1. B.D., 4 years, black, healthy from her birth until she was suddenly taken ill, on January 13, at 4 a.m. Convulsions, but no vomiting. Death at about about 7 a.m.

Post-mortem examination (with Dr. Earle, May Pen, at 6 p.m., January 13).

Spinal puncture gives about twenty drops of clear fluid. Diffuse hyperaemia of pia cerebralis, mostly pronounced on some parts of the convex surface. Slight oedema.

The *stomach* contains alimentary liquid without blood, and with no excess of mucus. Considerable hyperaemia and a few minute haemorrhages in the mucosa.

Diagnoses : Hyperaemia et oedema l.g. meningum cerebralium. Lympho-adenitis mesenterialis simplex. Hyperaemia et haemorrhagiae renum minimae.

Hyperaemia et petechiae mucosae ventriculi. Enteritis follicularis l.g. Helminthiasis intestinalis (Ascaris lumbricoides). Metamorphosis adiposa hepatis l.g.

Histological examination :—

Lymph nodules : Slight increase of stroma.

Liver : Patches of hyperaemia are seen in various parts of the sections ; the stroma around the portal vessels and the capsule are slightly increased in thickness and there is a beginning microcellular infiltration. The parenchyma is well preserved.

Kidney : Patches of hyperaemia are seen in various parts of the sections, and the stroma is slightly increased.

Suprarenal capsules : Considerable hyperaemia and marked vacuolization of cells.

Microbiological examination :—

Nasgar tubes inoculated with *spinal fluid* show growth of Gram-negative diplococci which change glucose, maltose and galactose, but not saccharose and lactose when examined in Jamaica, but in Liverpool they give different reactions which will be mentioned later on in the general discussion.

Smears from *brain*, *spleen*, and *liver* contain various bacilli and cocci.

Case 8. H.S., 3½ years, black, Post Road, by Chapelton. (Seen after death with Dr. Thomson). The child was, according to the mother, healthy until January 18, 1913, at 5 a.m., when she started vomiting and continued vomiting until death took place two hours later. No convulsions. The previous day she had eaten some unripe yam ; the mother partook of the same and had some vomiting immediately afterwards.

Post-mortem examination :—

The *stomach* contains alimentary fluid and a small amount of mucus. No haemorrhage ; no hyperaemia.

Duodenum : Slightly hyperaemic.

Diagnoses : Hyperaemia piae matris cerebralis l.g. Hypostasis pulmonum l.g. Enteritis follicularis l.g. Helminthiasis intestinalis (Ascaris lumbricoides). Lymphadenitis mesenterialis.

Microbiological examination :—

Nasgar tubes inoculated with *heart blood* remain sterile ; tubes inoculated with the sanguinolent *spinal fluid* show growth of Gram-negative diplococci which change dextrose, maltose, and to a very slight degree galactose, but not lactose, saccharose, and mannite.

Case 30. D.B., 9 months, black, Kingston Public Hospital, Dr. Thomson, January 31—February 1, 1913.

Sudden onset of vomiting and convulsions ; quite unconscious when admitted ; limbs rigid ; head retracted ; Kernig's sign marked in both legs, left more than right. No fluid obtained by lumbar puncture. Given 50 millions meningococcus-vaccine.

February 1. Rested fairly well during night. Only two attacks of convulsions no vomiting ; limbs still rigid. The child appears conscious. Temperature normal. Slight nystagmus. Slight internal strabismus, of left eye.

Post-mortem examination (performed by Dr. Thomson, who preserved the organs for me, as I was absent in the country).

Meninges congested. Lateral ventricles distended with blood-stained fluid. Upper part of spinal pia dark pigmented.

Internal organs normal.

Anatomical diagnosis : Hyperaemia meningum.

Bacteriological examination :—

Nasgar tubes inoculated with *spinal fluid* show growth of Gram-negative diplococci which change glucose, maltose and galactose, but not saccharose, lactose and mannite.

Case 33. E.B., 2 years, black, Rock, near Four Paths, with Dr. E. R. C. Earle.

Has been slightly ill for about three months. On February 3 he was unable to get up, ' he could not drink, became stiff and died at 11 a.m.' Vomiting not mentioned, nor convulsions.

Post-mortem examination : No jaundice.

Lumbar puncture gives clear, translucent fluid. Spinal cord not examined. Dura mater cerebralis normal ; pia shows moderate hyperaemia, mostly pronounced on the posterior portion of the convex surface ; no hyperaemia on the basis of the brain, but a small collection of slightly turbid fluid. The lateral ventricles are not dilated. The brain substance is of normal appearance.

Anatomical diagnoses : Hyperaemia meningum et mucosae ventriculi. Petechiae pericardii. Anaemia organorum. Helminthiasis intestinalis. Tuberculosis caseosa nodulorum lymphaticorum bronchialium.

Bacteriological examination :—

Spinal fluid, nasgar tubes : Gram-negative diplococci which change glucose, maltose and galactose, but not lactose, saccharose and mannite.

Case 43. C.B., 2 years, black, Old Hope, The Quarry, with Dr. C. R. Edwards, Half-Way Tree.

The child seems to have had slight cold for about two hours, then became very ill, vomited and died in convulsions at 5 a.m., on February 14, 1913.

Post-mortem examination, at 3 p.m., on February 14, about ten hours after death. Body fairly well nourished. No jaundice.

Lymph nodules : Axillary slightly enlarged, moderately hyperaemic ; inguinal considerably enlarged, slightly hyperaemic ; cervical moderately enlarged and hyperaemic.

Spinal puncture gives about 5 c.c. of clear, afterwards slightly turbid fluid. Spinal and cerebral durae normal, piae slightly hyperaemic, particularly on the convex surface of the brain. No exudation. Brain and cord normal. Lateral ventricles not distended.

Mesenteric lymph nodules are very much enlarged, and show punctate hyperaemia.

Spleen : 8 × 4 × 2 cms. Capsule normal, tissue normal, pink.

Anatomical diagnoses : Poly-lymphadenitis acuta l.g. Lymphadenitis mesenterialis m.g. Hyperaemia meningum. Sequelae pleuritidis sinistrae. Infarctus haemorrhagicus pulmonis dextri. Bronchitis. Enteritis l.g. Hyperaemia mucosae ventriculi

Histological examination :—

Axillary lymph nodules : Intense hyperaemia and infiltrating haemorrhages.

Mesenteric lymph nodules : Moderate hyperaemia.

Kidney : Irregularly distributed patches of hyperaemia.

Pancreas : Oedema of stroma.

Microbiological examination :—

Nasgar tubes of *spinal fluid :* Gram-negative diplococci and Gram-negative cocco-bacilli. The diplococci are isolated and are found to change glucose and maltose, but not galactose, lactose and mannite. Also the examination of the centrifugalized deposit shows Gram-negative diplococci and several other bacteria.

Case 55. A.T., 6 years, black, Barbican, St. Andrew, sent to hospital by Dr. Crosswell, Public Hospital, Kingston, March 2, 1913.

The child started vomiting on the evening of February 28, 1913, has been vomiting since then, the vomited substance being yellow. Convulsions began at 7.30 p.m. on the following day. When seen by Dr. Crosswell, all limbs were relaxed, there was no retraction of head, nor of abdominal wall; Kernig's symptom; pupils sluggish, equal; the child was semi-comatose, but could be roused, and could sit up; asked for water. He was immediately sent to hospital. On his arrival at the hospital he was moribund; the limbs were jerking, the head drawn back; temperature subnormal; skin cold and clammy; pupils dilated.

He was admitted at 8.30 p.m., and died at 9 p.m., on March 2.

Post-mortem examination, at 11 a.m., on March 3.

Lumbar puncture gives about 10 c.c. of clear fluid. Spinal meninges normal, without any hyperaemia; cord normal. Cerebral dura slightly hyperaemic; pia shows intense hyperaemia and some oedema on convex surface, slight hyperaemia on cerebellum, no hyperaemia at the basis. The lateral ventricles contain a small amount of slightly turbid fluid. The hemispheres show slight hyperaemia of the brain substance which is otherwise normal.

The *cervical and inguinal lymph nodules* are considerably enlarged and slightly hyperaemic, the *axillary nodules* considerably enlarged and intensely hyperaemic.

The *pericardium* contains a few drops of clear fluid; the epicard shows numerous petechiae and small ecchymoses on both the posterior and anterior surfaces of left ventricle, and to a less degree on the anterior surface of the right ventricle.

The *stomach* shows considerable capillary hyperaemia, corresponding to the whole length of the lesser curvature and neighbouring portions, and a few petechiae in cardiac portion; slight follicular swelling.

Duodenum slightly hyperaemic. Jejunum shows patches of capillary hyperaemia; no follicular swelling. Ileum shows hyperaemia and swelling of some of the lower Peyer's patches and considerable swelling of the solitary follicles in the last portion. Appendix 6 cms., normal. Colon normal. No parasites.

Mesenteric lymph nodules are slightly enlarged, pale, of normal consistence.

Tonsils: There is a superficial necrosis, diffuse microcellular infiltration into the connective and muscular tissues; the vessels are enormously distended.

Urine: Yellow, slightly cloudy, acid, contains no albumen, but gives a faint reaction for nucleo-albumen, it contains no sugar, no bile pigment, and no casts.

Anatomical diagnoses: Hyperaemia meningum et cerebri. Lymphadenitis multiplex. Hyperaemia et oedema pulmonum. Metamorphosis adiposa hepatis et pancreatis. Nephritis parenchymatosa acuta duplex. Ecchymoses epicardii et lienis. Hyperaemia ventriculi c. petechiis. Enteritis follicularis.

Histological examination:—

Axillary lymph nodules: There is moderate diffuse hyperaemia and some perivascular blood-infiltration.

Myocard: Sections of fragments fixed in Flemming's fluid show minute fat droplets in the muscle fibres.

Spleen: Diffuse hyperaemia.

Suprarenal capsule: Intense diffuse hyperaemia of the organ and of the surrounding connective and fatty tissues.

Kidney: Irregularly distributed patches of intense hyperaemia; the cells of the convoluted tubules are swollen and granular, with vacuoles near the basal membranes. There is a slight increase of the stroma, accompanied by a slight microcellular infiltration.

Liver : Considerable capillary hyperaemia, slight microcellular infiltration of stroma and penetrating amongst the liver cells, which show advanced fatty change mostly marked in the portal and intermedial zones.

Stomach : The sections show the transition from the oesophagus to the stomach. The oesophageal epithelium is fairly well preserved. The gastric epithelium on the contrary is to a very large extent destroyed and in some place completely destroyed. There is an intense hyperaemia, and in the gastric mucosa also minute haemorrhages. The gastric lymphoid follicles are enlarged and contain numerous polymorphonuclear leucocytes and there is also diffuse microcellular infiltration of both mucosa and submucosa of the stomach, whilst the same phenomena are but slightly pronounced in the oesophagus. There is some oedema of the stroma. Numerous 'mast-zellen' in all coats, especially of the oesophagus. Bacteria are exceedingly numerous in the superficial portions of the stomach, and in some places groups of bacteria are observed in the deeper layers in blood vessels.

Centrifugalization-deposit of the *spinal fluid* shows numerous endothelial cells, few lymphocytes and a few polymorphonuclear leucocytes.

Microbiological examination :—

Spinal fluid, cultures on nasgar : Gram-negative diplococci which change glucose, maltose and galactose, but not saccharose, lactose and mannite. No micro-organisms are seen in smears from spinal fluid and blood from lateral ventricles.

Group C contains cases resembling those in the two preceding groups in their clinical and anatomical characteristics, but no bacteriological examination of the cerebro-spinal fluid has taken place. In fact, the cases in the three groups, A, B and C, are often so much alike that the only constant difference is that in B meningococci have been demonstrated, in C they have not been examined for, and in A they have not been found, though carefully examined for. A satisfactory classification of the cases in this group cannot be attempted until the Groups A and B have been discussed. Group C contains 11 cases, namely, Cases 2, 5, 10, 11, 13, 18, 24, 25, 27, 32 and 48.

The cases in this group are given in abstracts only.

Case 2. R.B., 4 years, coloured (seen with Dr. Earle, May Pen), January 13. Lives in the house next door to the one in which Case 1 died, and was reported ill whilst the autopsy on this case was being performed. The child started vomiting at 7 p.m., and was seen at 9 p.m., when it was in a state of slight collapse, with soft and somewhat irregular pulse, about 90. It was stimulated with ether, and improved rapidly. Recovery.

This case was, according to Dr. Earle, one of so-called 'vomiting sickness.'

Case 5. A.A.J., 23 months, black, male, not seen by physician before death. Melrose.

Taken ill suddenly on January 15, at 5 a.m., with pain in stomach, and efforts of vomiting, but no actual vomiting. No convulsions. Death took place at 7 a.m.

Post-mortem examination, with Dr. Cooke, Mandeville.

The *kidney* measures 6 × 3½ × 1½ cms. Capsule easily detached. Surface smooth, with minute haemorrhages and considerable hyperaemia. Renal substance reddish grey, almost uniform throughout cortex and pyramids. A few minute haemorrhages and yellowish patches in the substance.

Liver: 16 × 11 × 4 cms. Capsule smooth and transparent. Substance uniformly greyish red, of normal consistence. No yellow, or soft patches.

Pancreas of normal size, colour, and consistence.

Oesophagus, *stomach*, and *duodenum* without pathological changes. No hyperaemia except very slightly in the duodenum. The stomach contains a small quantity of liquid food; no blood.

Jejunum of normal characters, except for a slight enlargement of the solitary follicles in its upper part. A few of the last Peyer's patches in the *ileum* are slightly enlarged.

Appendix, *coecum*, and *colon* normal. No intestinal parasites.

Mesenteric lymph nodules not enlarged.

Diagnoses: Hyperaemia meningum l.g. Tonsillitis chronica. Nephritis acuta l.g. Enteritis follicularis l.g. Splenitis acuta l.g.

Case 10. S.S., 4 years, brown, 4 Chancery Lane, seen with Drs. Dryden and Thomson in the Public Hospital, Kingston. January 21, 1913.

Admitted with her playmate, Case 9 (group A). Symptoms and course similar, but weakness less pronounced.

Microscopical examination of the *blood* shows no parasites.

Case 11. A.W., 2 months, black, 82 Ruin Lane, seen in Public Hospital, Kingston, with Dr. Thomson. Admitted January 21, 1913.

Admitted to hospital on account of continuous vomiting and collapse; has been ailing for one day. Breast-fed. No convulsions, and no other meningeal symptoms. Temperature 100° F. in rectum. Gums swollen and tender.

The following days no vomiting, and no other symptoms. Temperature normal.

Microscopical examination of the *blood* shows no parasites.

Case 13. E.S., 16 years, black, Four Roads (seen with Dr. Croswell). The patient was well until the morning of January 23, when she was taken ill, feeling unwell and vomiting twice during the day. She was not feverish. The following morning she felt very weak, and was taken to Dr. C's surgery where she vomited once. This vomit was watery, frothy, but those of the previous day were described as 'bilious.' The temperature was normal, the pulse somewhat weak. Dr. C. called me to see the patient, and I saw her with Dr. C. at noon. She looked normal, slightly drowsy. Pulse 92, slightly soft and irregular. No jaundice. Heart, liver and spleen-dullness normal. No abdominal pain, tenderness or contraction. No stiffness of neck or other muscles. No Kernig's symptom. No deviation of eyes. Numerous rhagadae on the lips, and a herpes-like eruption on the chin.

No parasites were found in blood smears.

(This case was considered by Dr. Croswell to correspond fairly well to the picture of 'vomiting sickness').

In the following case there were several attacks of 'meningismus,' during the course of a suppurative infection. When the child died, the possibility of a purulent meningitis was therefore considered, but both the post-mortem findings and the final part of the illness were in accordance with the type of vomiting sickness.

Case 18. A.B., 4 months, black, Bloxburgh, St. Andrew. Seen at Kingston Public Hospital, with Dr. C. A. A. Thomson. January 4—29, 1913.

January 4, admitted, with abscess of axilla, observed three weeks previously; is said to have had convulsions recently. No vomiting, nor diarrhoea. Abscess incised. Temperature normal.

January 14. Has had several attacks of convulsions. No rigidity; no Kernig's symptom. Bowels free. Incision wound healing.

January 17. No convulsions for the past 48 hours. Wound healed, but there is some hard infiltration of the surrounding parts. Temperature normal.

January 29. Return of convulsions yesterday, one lasting several minutes followed by exhaustion and death.

Clinical diagnosis: Posterior basal meningitis.

Post-mortem examination: (29 January, 1913).

Anatomical diagnoses: Atrophia universalis. Anaemia organorum. Hyperaemia piae matris cerebralis. Pediculosis capitis.

Case 24. M.E., Craigmill, black, adult, seen with Dr. George.

The patient had complained of cardialgia and nausea for several days. On January 29, she started vomiting at about 2 p.m.; she had no fever. She went on vomiting for two days, bringing up considerable, according to description enormous, quantities of watery, yellow or greenish fluid. Temperature normal. Pulse 92, regular, weak. No meningeal symptoms.

February 1. Patient admitted to hospital. No vomiting. Pulse and general condition improved, after tinct. strophanti.

Case 25. R.M., 10 years, black, Craigmill. Seen on January 31, 1913, with Dr. George.

Patient has vomited four times since morning; was seen by Dr. George at 4 p.m. There is some epigastric pain and tenderness, when he is seen by Dr. G. and myself at 11.30 p.m. Pulse fairly good, but not strong. No vomiting since this afternoon. No meningeal symptoms.

February 1. Improved.

Recovered.

Case 32. E.B., 6 years, black, Rock near Four Paths, with Dr. Earle. 'Quite well on the morning of February 3. In the afternoon she vomited greenish substance and went to bed. About midnight she again started vomiting and continued till daybreak of the 4th. The jaw was locked, she could not swallow, became stiff and ground her teeth.'

The patient died at 10 a.m., on February 4, 1913.

Post-mortem examination :—

Liver: 18 × 10 × 4 cms. Capsule normal; surface and substance uniformly reddish grey.

The *stomach* contains clear alimentary fluid; the mucosa shows, on both sides of the lesser curvature capillary hyperaemia and numerous petechiae.

The *jejunum* and *ileum* contain numerous ascarides; no other intestinal parasites. The Peyer's patches in the lower part of ileum are slightly enlarged and hyperaemic.

The *pancreas* is normal.

Anatomical diagnoses: Hyperaemia piae matris cerebralis. Hyperaemia et petechiae ventriculi. Enteritis l.g. Nephritis parenchymatosa acuta l.g. Helminthiasis intestinalis.

Case 48. L.W., 12 years, black, Windward Road, Doncaster Pen. Seen at Public Hospital, Kingston, with Dr. Thomson.

Admitted, on February 18, 1913, with history of sudden onset of vomiting the previous night. An aunt (case 47) and her infant were similarly attacked, the infant dying at home.*

Temperature on admission normal, and remained so. Patient drowsy. No vomiting. No muscular rigidity; no Kernig's symptom.

February 19, rested well last night. No malarial parasites in blood.

Patient recovered, and left Hospital on February 21; he was then apparently quite well, but the pulse was very frequent, 100—120, weak and slightly irregular.

Case 51. E.E.E., 20 years, black, seen with Dr. Croswell, on February 23; post-mortem examination with Dr. Edwards on February 24.

The patient was taken ill suddenly, on February 22, at 7 a.m. with convulsions, was seen by Dr. C. in the afternoon, was then very violent; after chloral and bromide she seemed to improve, though she had repeated convulsions. The following day, just after noon, she became unconscious, and comatose, and was in a dying condition, when seen on February 23, at 4 p.m. by Dr. C. and myself. Temperature was then 105° F., pulse could not be felt; she reacted a little, when given injections of ether and digitalin, so that the pulse could just be felt, filiform, at the radials, but not counted; the heart beats were weak, about 150 to 180. She died in coma at about 5 p.m.

When seen just before death, the pupils were of normal size; there was no retraction of the head, but slight rigidity of the neck-muscles. No Kernig's sign. Abdomen natural, no enlargement of liver, or spleen. Duration of illness: 34 hours.

Post-mortem examination, at 12 noon, on February 24, about 19 hours after death.

Body well nourished, healthy looking. Excoriations on the lips. Rigor present. No jaundice.

Cervical, inguinal and particularly the *axillary lymph nodules* considerably enlarged and intensely hyperaemic.

No spinal fluid obtained by lumbar puncture. Cerebral dura and pia without hyperaemia and exudation; lateral ventricles not distended; brain substance normal. Spinal dura, pia and cord perfectly normal in dorsal portion, the only part examined.

Hypophysis cerebri: Normal.

Pleurae: Left pleura shows numerous small ecchymoses, especially on the inferior lobe.

The *pericardium* contains a few c.c. of clear fluid; the epicard shows diffuse hyperaemia and numerous small ecchymoses on the posterior surface of the left

* A post-mortem examination was made on the infant by Dr. Gifford who informed me that he had found no hyperaemia of meninges and no pronounced lesions of any organ.

ventricle and auricle. Heart conus 9 × 10 × 5 cms.; apex is formed by left ventricle which is somewhat flabby. Endocard and valves normal. Myocard pale, with yellowish patches, of rather soft consistence.

Mesenteric lymph nodules slightly enlarged and hyperaemic.

Spleen: 10 × 8 × 2 cms. Capsule slightly thickened, whitish; colour of tissue normal; follicles not prominent, but a considerable number of small (from miliar to lenticular) yellow nodules are seen in the pulpa. Consistence normal.

Suprarenal capsules normal.

Left *kidney:* 12 × 6 × 3 cms. Capsule slightly adherent; surface smooth, except along some entering vessels, where capsule is adherent; surface pale yellowish, with patches of capillary hyperaemia; cortex pale, yellowish, with prominent glomeruli and some hyperaemic stripes; columnae Bertini still more yellow and rather soft; their tissue is bulging forward on the cut surface; the pyramids show slight hyperaemia of their bases.

Right *kidney:* 12 × 7 × 3 cms. Shows same characters as left.

Renal pelves, ureters and bladder hyperaemic. The calyces, pelvis and ureter on the right side contain a yellowish, purulent fluid. The bladder contains about 100 c.c. of pale, slightly turbid urine.

Both *ovaries* are hyperaemic; the right contains a fresh corpus luteum and the left also one, apparently of recent date. Both contain, besides, small serous cysts. The Fallopian tubes are not distended; only the left is patent; its mucosa is apparently normal, but the ovarian extremity contains purulent fluid. Uterus hyperaemic; its cavity measures, from orificium internum to fundus 4 cms.; collum 3 cms. Endometrium corporis hyperaemic and covered with slight purulent secretion. The cervix contains muco-pus; its mucosa is swollen, with minute haemorrhages. The portio vaginalis is discoloured, dark, with erosions.

Liver: 30 × 16 × 8 cms. Capsule normal; surface spotted red and yellowish grey, with subcapsular hyperaemia and minute haemorrhages; the tissue is uniformly yellowish grey; structure enhanced by slight hyperaemia of portal veins; consistence soft and extremely friable.

Pancreas of normal size, considerably hyperaemic and very soft.

Tonsils show small purulent particles on the surface, not enlarged.

Oesophagus: Slightly hyperaemic, contains greenish black fluid.

Stomach considerably distended, contains greenish black fluid with small black particles; no smell of phosphorus. The mucosa shows considerable hyperaemia of the cardiac portion, on both anterior and posterior walls, and a large number of petechiae; on the remaining parts of the mucosa a few small erosions are seen.

Duodenum shows hyperaemia and some petechiae.

Appendix 17 cms., normal.

Bile ducts and gall bladder normal; the former are patent, and the latter contains a moderate quantity of greenish bile.

Urine: Turbid; acid, contains about 1 per mille albumen and a few granular casts, no indican, no bile pigments and no sugar.

Anatomical diagnoses: Poly-lymphadenitis acuta. Haemorrhagiae pleurae sinistrae, pericardii, et capsulae hepatis. Hypostasis pulmonum. Laryngo-tracheo-bronchitis catarrhalis. Struma simplex l.g. Metamorphosis adiposa myocardii et hepatis m.g. Pancreatitis parenchymatosa acuta. Nephritis parenchymatosa acuta duplex m.g. (in chronica). Pyelo-ureteritis dextra. Endometritis. Salpingitis. Hyperaemia ovariorum. Noduli lienis. Gastritis ecchymotica cum erosionibus. Duodenitis ecchymotica. Hyperaemia jejuni et coli. Pharyngitis acuta.

Histological examination :—

Axillary lymph nodule : Intense hyperaemia and haemorrhages, both arteries, veins and capillaries being enormously distended ; there is diffuse erythrocyte infiltration of the tissues especially in the central portion of the nodule.

Liver : Intense fatty change and slight necrobiotic changes equally distributed throughout the lobuli ; several patches of capillary hyperaemia.

Pancreas : In the sublimate-alcohol specimens, only slight localized necrobiotic changes are observed, but in the Flemming specimens there is marked diffuse fatty metamorphosis present, all or nearly all cells containing minute fat droplets.

Kidney : The glomeruli are distended and some of them hyperaemic. Most of the epithelial cells in the convoluted tubules contain large vacuoles (fat, Flemming). A few tubules show marked necrobiotic changes of the cells. The vacuoles, when not filling the cell entirely, always occupy the basal portion of the cell ; as a rule the cell contains only one or a few fat drops. In some portions of the kidney the necrobiotic changes are more marked and the fatty metamorphosis less marked. In these portions there is some capillary hyperaemia. There is a slight increase of the connective tissue throughout the sections ; there is no microcellular infiltration.

Microbiological examination :—

No growth in nasgar and haemoglobin agar tubes inoculated with heart blood, except some colonies of large bacteria, probably accidental infection. No parasites in smears of peripheral blood and heart blood. No gonococci in smears from the right renal pelvis and endometrium.

In group D we include the cases in which the usual characteristics of meningococcus-meningitis were found, namely definite inflammatory changes in the meninges and meningococci in the cerebrospinal fluid. The following five cases fulfil these requirements : 4, 19, 29, 31 and 39. All these cases are given in abstract only.

Case 4. V.C., 2 years, female, black, of Craigmill, Portland, seen, after death, with Dr. H. J. George, of Buff Bay.

The child was taken ill at 5 p.m. on January 13, having been in good health previously. She had never before suffered from convulsions, fever, or vomiting. She started vomiting and continued vomiting, apparently without any effort, until her death which took place at about 5.30 p.m., i.e., about half an hour from the onset. The child did not appear feverish, there were no convulsions, and no stiffness of neck or other muscles was noticed.

Post-mortem examination was made at 6 p.m., on January 14, i.e., 25 hours p.m. Well nourished child. No jaundice.

Spinal puncture gives about twenty drops of clear, yellowish fluid.

Meninges : Intense congestion of both cerebral and spinal dura and pia, but mostly pronounced in the pia on the convex surface of the brain, and on the upper surface of cerebellum. Slight oedema of pia. Beginning sero-fibrinous exudation in median and Sylvian fissures.

Brain and spinal cord show, in some parts, irregularly distributed slight hyperaemia, but no other macroscopical lesions.

Mouth, *pharynx*, *tongue*, *tonsils*, *larynx*, *trachea* and *bronchi*, *thyroid body* and *thymus* are macroscopically normal. No liquid and no adhesions in *pleurae*.

Oesophagus, *stomach* and *duodenum* without pathological changes.

Liver : 17 × 10 × 4 cms. Capsule smooth, transparent. Colour slightly irregular, greyish with red patches. No yellow colouring except a few small patches in the left lobule. Substance slightly pale, otherwise normal.

Pancreas : Of normal size, colour and consistence.

Anatomical Diagnoses : Hyperaemia diffusa meningum. Leptomeningitis cerebralis fibrinosa incipiens. Hypostasis pulmonum l.g. Infarcti haemorrhagici pulmonis sinistri. Anaemia myocardii. Anthracosis lymphonodulorum bronchialium (et tuberculosis ?) Cystis haemorrhagicus ovarii dextri. Lympho-adenitis mesenterialis simplex. Enteritis follicularis l.g. Helminthiasis intestinalis (Ascaris lumbricoides).

Microbiological examination :—

Nasgar tubes, *spinal fluid :* Gram-negative diplococci which change glucose and maltose, but not galactose, lactose and saccharose.

Case 19. A.H., 4 years, black, 12 Beeston Street, Kingston. At Kingston Public Hospital (Dr. Thomson), January 18, 1913

Apparently well till January 17, at 11 p.m., when he started vomiting ; he had convulsions, was stiff at times ; the head was drawn back.

He was admitted to hospital early the following morning, and died at 8.30 a.m., on January 18.

Kernig's sign was present on both sides.

Lumbar puncture gave spinal fluid under considerable pressure ; the fluid was clear.

The urine was clear, contained no albumin, and no casts.

Post-mortem diagnoses (Dr. Scott) : Hyperaemia piae matris. Meningitis sero-fibrinosa. Sequelae pleuritidis sinistrae. Lymphadenitis mesenterialis. Metamorphosis adiposa hepatis, l.g.

Microbiological examination :—

Nasgar tubes, *spinal fluid :* Gram-negative diplococci which change glucose, maltose and galactose, but not lactose, saccharose, and mannite.

Case 29. B.W., 12 years, male, black, admitted to Kingston Hospital, January 25, 1913.

History of 'fever' ; delirium on admission ; temperature 100° F. ; no retraction ; no rigidity ; no Kernig ; spleen not felt ; passes urine in bed ; illness said to have lasted a week or longer. Diagnosis of malaria ; blood shows crescents. January 26 :—temperature 101° F., still unconscious, had two attacks of convulsions during night. Died January 28, 9.20 a.m.

Post-mortem examination was made at 2 p.m. (by Dr. Scott), on January 28, i.e., 5 hours p.m.

Thoracic organs : Nothing abnormal.

Spleen is dark, greyish-black, not much enlarged. 5 × 3 × 1 cms.

Liver is dark, greyish-black.

Congestion of *Pia* and surface of Brain is very marked ; thickish lymph over cortex, with purulent patches between hemispheres ; at base, greenish-yellow pus and abundant turbid fluid. Ventricles distended, about 5—6 ozs. of slightly turbid fluid in each. Spinal fluid turbid and abundant.

Anatomical Diagnoses : Leptomeningitis, fibrino-purulenta. Splenitis acuta Hepatitis parenchymatosa.

Microbiological examination :—

Spinal fluid : Smears show numerous intracellular diplococci ; cultures on nasgar show colonies of Gram-negative diplococci which change glucose, maltose and galactose, but not lactose, saccharose and mannite.

Case 31. H.M., 19 months, black. At Kingston Public Hospital, February 3—5,1913.

Admitted unconscious, on February 3, at 3.45 p.m. History of vomiting and convulsions of sudden onset.

Temperature 98. Slight strabismus. Limbs rigid. Lumbar puncture: no fluid. February 4, Temperature 102. Has vomited once. Head retracted. Kernig's sign doubtful. Knee-jerks not obtained.

February 5, very weak. Cheyne-Stokes respiration. Death.

Post-mortem examination (performed by Dr. Scott, who preserved the organs for me, as I was out in the country).

No congestion of meninges and brain, but lateral ventricles distended with turbid fluid. Inflammatory lymph at basis of brain. Spinal canal shut off by a layer of lymph like a diaphragm (neg. res. of lumbar punct. !) Internal organs normal.

Anatomical Diagnosis : Meningitis.

Microbiological examination :—

Nasgar tubes, *spinal fluid :* Gram-negative diplococci which change glucose, maltose and galactose, but not saccharose, lactose and mannite.

Case 39. U.M., 8 years, black, Osborne Store, Four Paths, Clarendon, with Dr. E. R. C. Earle, May Pen.

The child was healthy until Sunday, February 9, 1913, when he was taken ill with vomiting, at about 9 a.m. He improved after mint-tea and whisky, but on the following night vomiting started again, at about 3.30 a.m. on February 10. He vomited repeatedly during the day. He was seen by Dr. Earle at about 5 p.m., but was then already in a dying condition. He died at 6 p.m.

Post-mortem examination :—

Cervical, axillary and inguinal lymph nodules slightly swollen.

Lumbar puncture gives clear fluid. Dura mater normal. Pia mater slightly hyperaemic on the convex surface of the brain, more pronouncedly so on the basis and especially on the cerebellum; it appears normal on the upper part of the spinal cord, but there is a slight hyperaemia of its lumbar portion. There is a small amount of slightly clouded, serous exudation at the basis of the brain, of which liquid smears are taken. Lateral ventricles of normal aspect, as well as the substance of brain and cord.

The *stomach* is somewhat dilated and contains a considerable quantity of fluid of the character of intestinal contents. The gastric mucosa is normal in its larger part, but shows in its central portion, on the anterior surface and close to the lesser curvature, a large hyperaemic patch with several petechiae and a few petechiae scattered about on the neighbouring parts. Near the pylorus, on the posterior wall, considerable follicular swelling is observed. The duodenum shows hyperaemia, ecchymoses and follicular swelling. The jejunum is hyperaemic, especially in its upper portion; no haemorrhages. The intestinal tract is otherwise normal; it contains no parasites.

Anatomical diagnoses : Leptomeningitis incipiens. Lymphadenitis simplex l.g. Hypostasis et oedema l.g. pulmonum. Bronchitis. Metamorphosis adiposa myocardii et hepatis. Gastritis ecchymotica et follicularis. Enteritis l.g. Splenitis et perisplenitis chronica. Pancreatitis parenchymatosa acuta. Nephritis parenchymatosa acuta l.g.

Urine : Faint albumin-reaction and hyaline casts.

Histological examination :—

Spleen : Oedema of stroma.

Myocard : Fragmentation.

Pancreas : Extensive necrobiotic changes, most nuclei remaining unstained, and the cell limits seldom being recognisable.

Stomach : The mucosa shows considerable hyperaemia and some superficial haemorrhages, and diffuse microcellular infiltration.

Smears of spinal fluid after centrifugalization show a number of lymphocytes and a few polymorphonuclear leucocytes.

Microbiological examination :—

Nasgar tubes, *spinal fluid :* Gram-negative diplococci which change glucose, maltose and galactose, but not lactose, saccharose and mannite. Same result on haemoglobin agar. *Heart blood :* Growth of Gram-negative bacteria which ferment all sugars tested.

In smears from *liver*, chains and groups of diplococci are observed, some of them being situated in mononuclear leucocytes. In smears of spinal fluid and heart blood no cocci and no protozoal organisms are observed.

Personally, I saw no cases of cerebro-spinal meningitis of an ordinary type, but Scott described (1913), as already mentioned, several such cases in connection with his investigations on ' vomiting sickness.' As a further proof that *Diplococcus meningitidis* occurred in Jamaica at the same time that ' vomiting sickness ' was prevalent, I give abstracts of two observations, for which I am indebted to Colonel Wilson, R.A.M.C.

Case I. This was a case in a Sergeant-Major who was taken ill on the 2nd January, 1913, and died on the 6th. Clinical symptoms of meningitis were present and there was a rise of temperature during the first days of the disease, with an intermission on the 3rd and 4th day and a terminal rise up to 107·2 just before death. The post-mortem examination showed an incipient meningitis, the cerebro-spinal fluid being turbid ' almost purulent.' The spinal fluid contained Gram-negative diplococci which did not grow on human Pleuritis serum.

Case II. This case occurred in the Chaplain of the Forces who was taken ill on the 15th January, 1913. He presented marked clinical symptoms of meningitis, and the disease lasted for about ten days. The patient recovered. Meningococci were not demonstrated in the spinal fluid but developed in cultures from the naso-pharynx.

Group E is composed of three cases, in which anatomically a commencing meningitis was present, but no meningococci were found. In Case 17 an adult woman died after an illness lasting for about 60 hours, during which she had presented symptoms fairly typical of ' vomiting sickness '; her five years' old child was taken ill the same day as the mother, and died within six hours. The child did not come under my observation, but I am indebted to Dr. Edwards for the notes which are given below. The other patient in this group, Case 35, was a girl of 11 years, living in the same house as Case 36,

a boy of 11. Both children were taken ill at the same time, with the same symptoms, and the duration of the illness was nearly the same in both cases. All these circumstances evidently point to the same disease having attacked the two children; in spite of all, the two cases must be classified under different headings, as the results of the post-mortem examinations, which were performed at the same time, differed considerably. In Case 35 there was a marked, though commencing inflammation of the meninges, but apparently no pathological changes in the stomach or intestines. In Case 36, on the other hand, there was a well marked haemorrhagic gastritis, whilst the meninges showed only slight diffuse hyperaemia, a common phenomenon in acute infectious diseases. Case 36 is therefore discussed as belonging to the following group of cases 'suspicious of yellow fever,' whilst Case 35 is considered, as it anatomically must be considered, as one of meningitis. In the latter case staphylococci were cultivated from the spinal fluid, but it appears extremely unlikely that this occasional occurrence of a very common organism should be of any etiological importance. All circumstances considered, it is probably correct to regard the two cases, 17 and 35, as meningitis of unknown origin, but belonging essentially to the same disease as is represented by the cases.

Thus, it may be that the separation of the two cases from those in Group A occurring in the same places and under identical circumstances is only provisional. Case 62 also somewhat resembles the type in Group A.

Case 17. M.H., 27 years, St. Andrews. Admitted January 29, 3.10 p.m. Public Hospital, Kingston, having vomited for the past 36 hours, complaining of headache and pain in the limbs.

Whilst in hospital, no vomiting occurred, but patient had some mucopurulent expectoration. Temperature 99°. Pulse about 80—90, regular and full to begin with, apparently with somewhat increased tension, but on the morning of January 30 it was weak and irregular. She was conscious during the first day, but the last morning she was in a state of quiet delirium and gradually passed into coma. There were no shrieks, but some grinding of the teeth. No muscular rigidity, and no Kernig's symptom. Knee-jerk slightly increased on the left side, but could not be produced on the right. No convulsions.

Spinal puncture made on January 30, about noon. Clear watery fluid comes out, rapidly dripping; about two c.c. taken.

Patient died at 2.15 p.m., January 30.

Post-mortem examination: (With Dr. Scott) at 3.30 p.m., on January 30.

Intense hyperaemia of *cerebral pia*, less pronounced of spinal pia. Some oedema and beginning fibrinous exudate over the cerebral hemisphere, not on the basis, nor on the spinal cord.

The *stomach* shows slight oedema of the mucosa, and in some parts slight capillary hyperaemia ; a pin head-sized superficial ulcer on the posterior surface, near the lesser curvature.

Anatomical diagnoses : Leptomeningitis cerebralis incipiens. Metamorphosis parenchymatosa et adiposa myocardii. Ulcus ventriculi parvum. Gastritis acuta l.g. Nephritis acuta parenchymatosa. Perisplenitis chronica fibrosa. Metamorphosis adiposa et parenchymatosa hepatis. Pancreatitis parenchymatosa acuta. Helminthiasis intestinalis.

Microbiological examination :—

Nasgar tubes, *spinal fluid :* remain sterile.

This patient was the mother of a child which was taken ill and died on January 28th, i.e., the same day that the mother's illness began. This case was observed by Dr. Edwards, to whom I am indebted for the following particulars, which show that the case, if classified, would have been included in Group A.

L.F., 4 years, 10 months, was taken ill at 4 p.m. Vomited several times convulsions set in and coma and death supervened at 10 p.m. Dr. Edwards performed the post-mortem examination and found :

Conjunctivae slightly jaundiced. Pleurae, lungs, heart, pericardium, peritoneum, liver and spleen normal. Kidneys pale, the stroma show slight conjugation of the cardiac portion, no blood in the lateral ventricles of the brain. Meninges not congested.

Nasgar tubes were inoculated at the post-mortem examination with the *spinal fluid*. They remained sterile.

Case 35. E.M., 11 years, black, Tollgate, with Dr. Earle. Living in same house as No. 36. May Pen.

The child was healthy until Tuesday, February 4, 1913, at about 3 p.m., when she started vomiting some black substance, first declared so by grandmother, who observed the child, but on further questioning she declared that the vomiting had been colourless ; she continued vomiting until the following afternoon, when the vomits ceased, and soon after convulsions, retraction of the head, and contraction of the jaw were observed. She died at 4 p.m., on the 5th, without medical attendance.

Post-mortem examination :—

Dura mater cerebralis normal. Pia mater shows slight diffuse hyperaemia and oedema, and beginning fibrino-purulent exudation around some of the vessels on the convex surface. Brain substance of normal appearance.

Anatomical diagnoses : Leptomeningitis cerebralis incipiens. Lymphoadenitis mesenterialis simplex. Splenitis acuta. Nephritis acuta. Pancreatitis parenchymatosa acuta. Helminthiasis intestinalis. Cadaverositas universalis.

Microbiological examination :—

Spinal fluid : Nasgar tubes, no growth of diplococci, but of Gram-positive staphylococci.

Case 62. R.A.L., 9 years, black, seen, after death, with Dr. C. R. Edwards, at Kingston Public Hospital, March 9, 1913.

Post-mortem examination :—

Lumbar puncture gives a few c.c. of clear fluid, with admixture of blood. Cerebral sinuses distended with blood clots ; pia considerably hyperaemic, especially on convex surface and with slight sero-fibrinous exudation at the basis. Lateral ventricles not distended, contain a very small quantity of serous fluid. Spinal dura normal ; pia very slightly hyperaemic in lumbar portion and slightly pigmented. Brain and cord normal.

Urine : Contains traces of albumin and nucleo-albumin.

Anatomical diagnoses : Leptomeningitis cerebralis incipiens. Splenitis acuta. Enteritis follicularis. Nephritis parenchymatosa acuta l.g. Petechiae epicardii. Infarcti haemorrhagici pulmonis dextri. Sequelae pleuritidis duplicis. Adenitis bronchialis caseosa. Endarteriitis aortae incipiens. Helminthiasis intestinalis. Pediculosis capitis.

Histological examination :

Cerebral basis : The pia mater shows microcellular infiltration, no organisms are observed.

Spleen : Considerable hyperaemia.

Liver : Moderate fatty change, oedema of stroma.

Pancreas : Both the alveolar epithelia and the islets of Langerhans of normal appearances. The latter in particular are very well preserved.

Kidney : The epithelia of the convoluted tubules show granulation metamorphosis, and slight necrobiosis, also slight vacuolization in the basal portions of many of the cells. In osmic acid preparations minute fat droplets are seen, chiefly in the basal portions of the epithelia of the convoluted tubules, but also in other parts of these cells and in other cells, the epithelia of Bowman's capsules and the glomerulus-cells also being attacked.

Duodenum : Considerable microcellular infiltration of the mucosa. The lymphoid follicles are considerably enlarged, numerous mast cells are present.

Lung infarct : alveoli filled with blood of apparently normal composition, slight epithelial desquamation and microcellular infiltration of stroma in neighbouring parts.

Microbiological examination :—

Nasgar tubes inoculated with *spinal fluid* and *heart blood* remain sterile.

Group F is very important. It contains cases in which one or several symptoms gave rise to a suspicion of yellow fever. The suspicious symptom which has been chiefly considered is black vomit. Petechiae in the gastric mucosa may easily occur in other conditions, and fatty liver is common in acute infections in children. Therefore, these two phenomena have not been considered sufficient to justify the inclusion of cases in this group, not even if supported by the presence of acute nephritis. All these lesions are so common in 'vomiting sickness,' that they must really be considered in the general discussion. On the other hand, dealing with extremely acute cases, the possibility must be admitted that jaundice may not have had time to develop. As it, moreover, is known that jaundice is not invariably present even in fatal cases of yellow fever, the

presence of this symptom was not considered necessary in order to include a case in this group. It is true, of course, that also black vomit is no absolutely constant symptom, but it would not be likely that it should be absent in rapidly developing and fatal cases. The observation of black stomach contents at the post-mortem examination is regarded as equivalent to the occurrence of black vomit. In this way, and considering the general characters, the following five cases were regarded as suspicious: Nos. 23, 36, 38, 47 and 54. Notes of all these cases are given below for reference. In Case 23 there is little doubt that yellow fever is the correct diagnosis. No complete clinical history could be obtained, as the patient, a Chinaman, was unable to answer questions when I saw him, and his Chinese friends explained themselves somewhat poorly in English. Thus we could not find out whether the patient had suffered from febrile symptoms, though it was evident that he had complained of severe headache. It was also ascertained beyond doubt that black vomit had been present, and the same could be concluded from the aspect of the tongue. Examination of the blood showed absence of malarial parasites, but a few intracorpuscular bodies were found of the type of *Paraplasma flavigenum*. The post-mortem findings were typical of yellow fever in the essential details; the small whitish patches which were found in the cerebral pia mater were possibly remnants of some old meningeal affection, but there was no fresh inflammation. The appearances of stomach, liver and kidney were those met with in yellow fever. The liver showed, microscopically, marked fatty and necrobiotic changes, in some places of the Rocha-Lima type.* No meningococci developed in the cultures, but diplococci were found in smears from various organs, an observation which will be referred to later on.

The anatomical findings in Case 36 are very suspicious of yellow fever, in spite of the absence of jaundice. Considering, however, that gastric and pericardial petechiae were frequently observed also in cases of 'vomiting sickness' which were not suggestive of yellow fever, I should not feel confident in making

* Rocha-Lima (1912) described a type of necrobiosis, combined with fatty change, in the liver, which he considers characteristic of yellow fever; the essential characteristic is that the necrobiotic changes are mostly pronounced in the intermediate portions of the lobules. This type is, according to my experience, common but not constant in yellow fever.

a diagnosis of yellow fever in such a case in the absence of a typical clinical history and of an examination of blood taken during life.

Case 38 is similar to Case 36, with the additional symptom of jaundice, thus strengthening the suspicion of yellow fever, but even thus the evidence cannot be regarded as conclusive.

The Cases 47 and 54 are also essentially similar. The symptoms are not suggestive of yellow fever, but rather of a meningeal affection, though not markedly so. In Case 54, however, black vomit was observed; in this case the peripheral blood was examined, and no *Paraplasma flavigenum* was found.

All these cases illustrate my previous statements about the difficulties which beset the post-mortem diagnosis of yellow fever. In all four cases the pathological lesions point to this diagnosis, but the clinical and epidemiological characters make one hesitate in pronouncing it, in the absence of conclusive parasitological evidence.

Case 23. C.C., male, about 20 years, Chinese, Charlestown. Seen, on January 31, 1913, with Dr. H. J. George.

The patient had lived in Jamaica from when he was a boy, and had been in good health up till present illness.

He felt ill on January 27, started vomiting at about 10 a.m., to begin with yellowish, later on greenish, and on January 30 and 31, red and black. He was seen by Dr. George on January 31, in the morning; the temperature was then normal. An injection of meningococcus-vaccine was given. Near midnight, on the same date, I saw the patient, together with Dr. George. He was then in a semi-comatose condition, not answering questions. Temperature was normal, and it was not quite clear whether he had been febrile, but it was stated that he had suffered from headache, besides the vomiting. Pulse about 90, weak, and slightly irregular. Respiration stertorous. No enlargement of spleen or liver. No abdominal tenderness. Perhaps slight jaundice of conjunctivae. The tongue has red borders, but is otherwise covered with a blackish fur.

On February 1, at 8.30 a.m., the patient arrived at the hospital in Buff Bay, in an extremely weak condition. The temperature was not taken, but the skin felt very cold to the touch. Pulse was filiform. There was no vomiting, but enema provoked a greenish-black evacuation. The patient did not react on stimulating treatment, and died at 11.30 a.m.

Post-mortem examination, at 2 p.m., 2½ hours after death.

Well nourished, fat body. Rigor present. Livores are very extensive on back and sides, and are also present on the front of the femora. Slight jaundice.

Spinal puncture gives blood-stained fluid. Spinal cord not examined.

Brain. Dura intensely hyperaemic, pia likewise hyperaemic, but less pronouncedly. No liquid exudate, but on the convex surface some whitish patches, in one place more yellowish. Brain preserved unopened, but right ventricle punctured and nasgar tube inoculated with point of needle.

Abdominal cavity contains no fluid. Peritoneum of normal aspect. Abdominal organs normally situated.

Spleen 13 × 11 × 2½ cms. Capsule smooth, transparent. Colour, structure, and consistence normal.

Kidneys 12 × 6½ × 3 cms. Capsule easily detached; Surface grey with a few dilated stellate veins. Cortex grey, with some yellowish patches. Pyramidal bases hyperaemic. Consistence normal.

Liver 30 × 15 × 7 cms. Surface yellowish grey, with hyperaemic patches. Capsule smooth, transparent. Cut surface uniformly yellowish grey. Consistence slightly diminished, pasty.

Pancreas of normal size and consistence, slightly hyperaemic.

The *stomach* contains a considerable amount of greenish fluid, and, adherent to the mucosa, black, coffee-ground like, mucous matter, with numerous blood-streaks. The mucosa shows considerable hyperaemia, chiefly of the posterior wall, and numerous petechiae, especially on both sides of the lesser curvature, and on the posterior wall. The duodenum contains greenish black fluid, and its mucosa is slightly hyperaemic. In the middle portion of the jejunum there is a small ecchymosis, and in the remaining portions of the bowel some hyperaemic patches are observed.

The *bladder* contains about 100 c.c. yellow, slightly turbid urine. Mucosa normal.

No liquid in *pleurae;* but adhesions over the superior lobe of the left lung.

The *lungs* show intense hyperaemia of postero-inferior portions; no oedema, no infiltrations, no infarcts. No definite signs of tuberculosis in lungs, or bronchial lymph-nodules.

Mucosa of trachea hyperaemic.

Thyroid body of normal aspect.

Pericardium contains no excess of fluid. No petechiae.

Pulmonary artery free.

Endocard and myocard of normal aspect.

Urine: Slightly turbid, acid, contains albumin; no sugar; faint reaction for bile-pigments; numerous hyaline and granular casts; a few leucocytes and erythrocytes; numerous epithelial cells.

Anatomical Diagnoses: Gastritis ecchymotica. Metamorphosis adiposa hepatis m.g. Pancreatitis parenchymatosa acuta. Nephritis parenchymatosa acuta. Hypostasis pulmonum m.g. Leptomeningitis cerebralis veteris dati l.g. Icterus universalis l.g.

Histological examination:—

Liver: Extremely intense vacuolization of cells (fat), as a rule most marked around portal vessels. Marked necrobiotic changes which are often most intense in the places of advanced fat changes around the portal vessels, but in other parts most intense in the intermediate zone. Considerable capillary hyperaemia, irregularly distributed. Some microcellular infiltration of the periportal connective tissue.

Pancreas: Hyperaemia and slight haemorrhagic infiltration; oedema of stroma. Marked necrobiotic changes, the cells in Langerhans's islets being as seriously affected as those in the parenchyma proper.

Kidney: Vacuolization of basal portions of cells (fat). Some increase of stroma which is oedematous; slight microcellular infiltration, considerable hyperaemia. Coagulated serum in Bowman's capsules around the retracted glomeruli.

Microbiological examination :—

Nasgar tubes, *spinal fluid :* Sterile. Nasgar tubes, ventricular fluid, Gram-negative bacteria, no diplococci. In smears of peripheral blood a few intracellular ring-shaped organisms are found of the type of *Paraplasma flavigenum.*

No protozoal organisms are found in smears of organs, but in smears from spleen and liver various bacteria are observed ; amongst these are diplococci which are Gram-negative and in several instances intracellular.

Case 36. P.B., 11 years, black, Tollgate, with Dr. Earle, May Pen. Living in the same house as No. 35.

The boy had been in poor health previously and often had ' fever.' He was taken ill on Tuesday, February 4, 1913, at about 3 p.m., when he began to vomit ; he vomited all night and during the next day, until he died near midnight on the 5th, without medical attendance. He had no convulsions, but retraction of the head was observed by the mother. During his previous illness he had never suffered from vomiting.

Post-mortem examination : At 8 p.m., on February 6, 20 hours after death. Poorly nourished body. Rigor present. No jaundice. No cutaneous affections or parasites.

No *spinal fluid* obtained by lumbar puncture ; spinal cord not examined. Dura mater cerebralis normal. Pia mater shows slight acute hyperaemia over all parts of the brain. No oedema or exudation. No distension of lateral ventricles ; a small quantity of clear fluid is seen at the basis of the skull. Brain substance shows normal aspect.

The *abdominal cavity* contains no fluid. The peritoneum is of normal aspect. The abdominal organs are normally situated.

The *mesenteric lymph nodules* are considerably enlarged and hyperaemic, of normal consistence.

The *gastric and intestinal vessels* are considerably distended. There are a few easily detached intussusceptions of the small bowel. Tongue coated with white fur. No haemorrhage of gums. Tonsils slightly enlarged ; no signs of acute inflammation. Oesophagus normal. The stomach contains brownish, apparently haemorrhagic fluid ; the mucosa is hyperaemic and shows on a small portion of the posterior surface, close to the lesser curvature numerous petechiae. The duodenum is hyperaemic, otherwise normal ; jejunum and ileum, show hyperaemic patches ; no haemorrhages, and no swelling of Peyer's patches, or solitary follicles ; no ulcers. No intestinal parasites.

Liver : 22 × 13 × 6 cms. Capsule smooth, transparent ; colour pale grey with yellowish patches ; consistence distinctly diminished. The duodenal papilla presents a small, prominent mucous mass which, however, gives way when pressure is exerted on the gall bladder, and the bile then flows out freely ; mucosa of bile ducts and gall bladder normal ; bile remarkably dark, and somewhat viscid.

Pancreas is hyperaemic, and of considerably diminished consistence.

Several *lymph nodules* at the porta hepatis are enlarged and show haemorrhagic infiltration.

Spleen : 19 × 5 × 1½ cms. Capsule smooth, slightly thickened ; tissue somewhat pale, with slightly prominent follicles.

Suprarenal capsules normal.

Left *kidney* 8 × 5 × 2 cms. Capsule easily detached ; surface is smooth, shows capillary hyperaemia, but no distension of stellate veins ; cortex pale yellowish ; pyramidal bases considerably hyperaemic. The right kidney presents the same characters as the left. Mucosa of renal pelves, ureters, and bladder normal.

The *pleurae* contain no fluid and show no adhesions. The lungs are pale, showing only a very slight hyperaemia of the posterior portions; the lung tissue contains air throughout; there are neither oedema, infiltrations, nor infarcts.

The *pericardium* contains a few drops of clear fluid. The visceral pericardium shows a considerable number of minute petechiae and small ecchymoses on the anterior, lateral, and posterior surface of the left ventricle, especially close to the auricle, and also a few similar ones on the auricle. Heart conus $7 \times 7 \times 2\frac{1}{2}$ cms. Endocard and valves normal. Myocard pale grey, with more yellowish patches; consistence distinctly diminished.

The *larynx* and *trachea* are normal; the smaller bronchi contain mucous fluid.

The *thyroid body* and the *hypophysis cerebri* are of normal size and aspect.

Anatomical diagnoses: Gastritis ecchymotica. Hyperaemia intestinorum. Metamorphosis adiposa hepatis. Pancreatitis parenchymatosa acuta. Nephritis parenchymatosa acuta. Lympho-adenitis mesenterialis et portae hepatis. Perisplenitis chronica l.g. Ecchymoses pericardii. Metamorphosis adiposa myocardii. Hypostasis pulmonum l.g.

Histological examination:—

Spleen: Hyperaemia; oedema of stroma.

Pancreas: Localized necrobiosis; oedema of stroma.

Kidney: Localized hyperaemia, granular change and karyolysis in many cells in the convoluted tubules. Some basal vacuolization.

Stomach: Some increase of lymphoid tissue and moderate diffuse microcellular infiltration; oedema of submucosa.

Duodenum: Hyperplasia of Brunner's glands and lymphoid tissue, slight microcellular infiltration. Necrobiotic changes in the mucosa.

Brain: Hyperaemia and slight superficial microcellular infiltration.

Case 38. D.L., 8 years, black, admitted to Kingston Hospital in a dying condition, on February 7.

The boy had been ill with fever and vomiting. No further particulars were obtained.

Post-mortem examination, on February 7, at 12.30 p.m.

Anatomical diagnoses: Icterus universalis l.g. Gastritis ecchymotica c. erosionibus. Metamorphosis adiposa hepatis. Nephritis parenchymatosa acuta c. metamorphosi adiposa. Metamorphosis adiposa myocardii. Hyperaemia piae matris cerebralis. Pancreatitis parenchymatosa acuta.

Black and blood-stained mucus is adherent to the wall of the stomach, which contains a few milk-clots.

A few slightly hyperaemic patches are seen in the small and large bowel. The Peyer's patches are likewise slightly hyperaemic.

Liver: 650 grms., yellowish grey, homogeneous, pasty.

Kidneys of normal size; capsule easily detached; surface congested, with yellow patches; cortex yellowish; consistence diminished.

Spleen of normal aspect. Slight diffuse hyperaemia of pia cerebralis, no odema or inflammatory exudate.

Urine: The bladder contains a considerable quantity of pale urine, which contains a small quantity of albumen, no sugar, no bile-pigments, few hyaline casts, numerous epithelial cells, a few erythrocytes and leucocytes.

Histological examination:

Lymph nodules: Hyperaemia and perinodular lymphoid infiltration.

Myocard: Numerous small vacuoles (fat) in muscle fibres.

Liver: Advanced fatty metamorphoses, all zones being equally affected. Marked anaemia.

Pancreas: Advanced necrobiotic changes, most nuclei being invisible and cell limits poorly defined.

Kidney: Large vacuoles (fat) occupying basal parts of cells in convoluted tubules. Most nuclei staining well, straight tubules not affected. Marked anaemia.

Medulla: Oedema and slight infiltration of pia.

Case 47. A.W., 20 years, black, Windward Road, Doncaster Pen. Seen in Public Hospital, Kingston, with Dr. Thomson.

Admitted in a weak condition. History of sudden onset of vomiting last night. No history of previous illness. Her infant, $1\frac{3}{4}$ years old, mentioned in note to case 48, was attacked earlier in the evening, and died. A nephew also started vomiting during the night (case 48).

Temperature 98·4° on admission. Pulse small and irregular. Patient complains of pain in the epigastrium.

Temperature went up in the afternoon to 99° and 100°, but in the evening again down to 98°, where it remained until the time of death. Heart's action was in the afternoon regular; sounds clear. Lungs clear. Tenderness in epigastrium. Spleen not felt. Pupils equal, not contracted, nor dilated, react to light and accommodation. No vomiting since admission, but drowsiness. No rigidity; Kernig's symptom not present.

February 19. During the afternoon patient's condition grew weaker; the drowsiness passed into coma. The breathing became laboured; the pulse was very small and irregular, but responded readily to stimulation. No rigidity of limbs.

At 4 a.m., patient appeared to be sinking; she was quite comatose, with Cheyne-Stokes's respiration; pulse small and irregular.

Lumbar puncture gave clear, watery fluid. Urine could not be collected.

Death at 5.35 a.m.

Post-mortem examination at 7 p.m., on February 19, 13 hours after death.

Body well nourished; rigor present; no jaundice. A few *Pediculi capitis*, no other parasites, nor affections of the skin.

Axillary *lymph nodules* moderately enlarged and hyperaemic; inguinal enlarged, cervical normal.

The subcutaneous fat shows, in various places, small haemorrhages.

Dura spinalis normal; pia hyperaemic, especially in lower part. Spinal cord normal. Cerebral dura normal; pia considerably hyperaemic, particularly on the convex surface. No exudation, but some haziness over the basis and over the Sylvian fissures. Lateral ventricles not distended, contain a few drops of clear fluid. Brain substance normal.

Hypophysis slightly enlarged, hyperaemic, and with soft, yellowish patches.

Pleurae normal. Lungs slightly hyperaemic in postero-inferior parts, contain a few minute haemorrhagic infarcts. Smaller and larger bronchi, trachea and larynx show diffuse hyperaemia and some mucous secretion. Bronchial lymph nodules not enlarged.

Pericardium contains a few c.c. of serous fluid; conus 9 × 11 × 5 cms.; epicard shows several small ecchymoses on posterior and left surface of left ventricle and auricle. Endocard and valves normal. Myocard shows small yellowish patches; consistence somewhat diminished; left ventricle fairly well contracted.

Thyroid body normal.

Peritoneum normal, without fluid. Abdominal organs normally situated.

Spleen: Small, 11 × 5 × 2 cms. Capsule normal; colour pale; follicles grey, slightly prominent.

Suprarenal capsules normal.

Mesenteric *lymph nodules* slightly enlarged, not hyperaemic.

Left *kidney:* 12 × 6 × 4. Capsule easily detached; surface extremely pale except in a few places where there is some dilatation of the stellate veins. Cortex pale, yellowish grey, and some of the septa completely yellow, of almost caseous aspect, the parenchyma structureless, bulging on the cut surface. Pyramids pale. Consistence of the whole organ considerably diminished, and of the yellow parts, soft, pasty. Right kidney 11 × 7 × 3 cms.; characters similar to those of the left. Mucosae of renal pelves, ureters and bladder normal. The bladder contains a small quantity of pale urine.

Liver: 29 × 15 × 8 cms. Capsule normal; surface pale, yellowish; tissue yellowish grey, homogeneous; consistence pasty.

Pancreas of normal size, but of somewhat diminished consistence.

Tongue covered with white fur. Tonsils normal.

The *stomach* contains a large quantity of reddish black fluid, with black particles, coffee-grounds like, also some blackish mucus (the fluid is acid, and gives reactions for haemoglobin); mucosa hyperaemic; no haemorrhages visible. Duodenum contains considerable quantity of dark, sanguinolent fluid, mucosa intensely hyperaemic, with minute petechiae. Jejunum normal. Ileum hyperaemic. Colon shows marked hyperaemia. Appendix 18 cms. Two ascarides in ileum.

Anatomical diagnoses: Hyperaemia meningum. Hypostasis et infarcti haemorrhagici parvi pulmonum. Ecchymoses pericardii. Metamorphosis adiposa myocardii. Ecchymoses telae adiposae subcutaneae. Gastritis acuta haemorrhagica. Duodenitis acuta haemorrhagica. Hyperaemia coli. Metamorphosis adiposa hepatis maximo gradu. Nephritis acuta duplex parenchymatosa c. metamorphosi adiposa m.g. Metamorphosis adiposa (?) pancreatis. Lymphadenitis axillaris, inguinalis et mesenterialis acuta. Pediculi capitis. Helminthiasis intestinalis.

Histological examination:—

Axillary lymph nodules: Hyperaemia.

Pituitary body: Hyperaemia.

Suprarenal capsule: Diffuse hyperaemia, especially intense in the periglandular tissues where also infiltrating haemorrhages are observed.

Liver: Intense fatty metamorphosis. Necrobiotic changes are present throughout the organ; in some places these phenomena show the Rocha-Lima type, but most often only insignificant remains of protoplasm are preserved in the cells surrounding the hepatic veins, where the affection in many places appears mostly advanced. There is slight microcellular infiltration of the periportal tissues.

Pancreas: Minute fat droplets are observed in the alveolar epithelia and slightly larger droplets in the cells of Langerhans's islets.

Kidney: The epithelia of the convoluted tubules show granulation of protoplasm and their basal portions are occupied by vacuoles which correspond to fat droplets in osmic acid specimens. These phenomena are present with some irregularity in nearly all the convoluted tubules and exceedingly intense in many of those of the first order. They are also very intense in the ascending limbs of Henle's loops, but there is hardly any fatty change of the cells in the descending limbs and collecting tubules. A number of patches of intense capillary hyperaemia

are observed and in the neighbourhood of these patches there is some microcellular infiltration. There is moderate increase of the stroma especially of the pyramids, the glomeruli are slightly retracted and minute fat droplets are observed in their cells.

Myocard : Moderate fatty change.

Microbiological examination :—

Spinal fluid, nasgar tubes : Gram-negative diplococci which change glucose, galactose and maltose, and saccharose slightly, but not lactose and mannite ; also some colonies of Gram-negative cocco-bacilli.

Heart blood, nasgar tubes : Gram-negative cocco-bacilli. Smears of peripheral blood and heart blood show no protozoa but in the heart blood some large bacteria.

No organisms are seen in smears from spinal fluid after centrifugalization.

Case 54. D.A., 7 years, coloured, Eastward Villa, Hagley Park. Seen, after death, with Dr. C. R. Edwards, Halfway Tree, on February 25 and 26, 1913.

The child has never been ill before. She felt slightly unwell on February 23, in the evening, was crying and complaining of pain in the stomach ; during the following night she woke up, at 1.30 a.m., February 24, groaning, and had three attacks of convulsions with clonic spasms. During the next day she vomited several times clear fluid, and did not take any food ; she did not seem to have fever, but, according to the mother, the skin turned yellow (vide infra). She was awake during the whole of the following night, restless, semi-unconscious, and complaining of abdominal pain. At 9 a.m., on February 25, she was brought to Dr. Edwards' office, where she remained until her death. Whilst here, several (8) convulsions were observed ; they consisted of clonic spasms, with final tonic contractions ; she vomited repeatedly, to begin with clear, watery fluid, but later on dark, coffee-grounds like matter, and reddish, thin fluid, with suspended small particles, in large quantities. She was unconscious, became gradually weaker, and died at about 4 p.m., just a quarter of an hour before my arrival.

When seen after death, the body appeared rather yellow, but there was no trace of yellow in the conjunctivae. Spinal fluid, blood, urine and vomited matter collected by Dr. Edwards.

Spinal fluid : About 20 c.c., clear.

Bladder contained only a few drops of pale urine.

Post-mortem examination, at 11 a.m., on February 26, 19 hours after death :—

Body somewhat poorly nourished. Rigor present. Extensive livid patches on the back, invading even the flanks. No jaundice of conjunctivae, but the body has a peculiar yellow colour. No skin affection, and no external parasites, except lice in the hair.

Spinal venous plexus gorged with blood. Dura spinalis normal, cerebralis slightly hyperaemic ; pia spinalis and cerebralis intensely hyperaemic, and the latter slightly oedematous at the basis. Lateral ventricles not distended, contain no fluid. Brain and cord normal.

Hypophysis softened, otherwise normal.

Inguinal, axillary and cervical lymph nodules considerably enlarged and intensely hyperaemic.

Left *kidney :* 8 × 5 × 2 cms. Capsule easily detached ; surface smooth, dark, with capillary hyperaemia, shows a few yellow patches ; cortex reddish grey ; glomeruli normal ; columnae Bertini pale, yellowish ; pyramidal bases hyperaemic ; consistence considerably diminished. Right kidney shows same characters as left.

Tonsils : Hyperaemic, contain several small pus foci.

Mucosa of pharynx swollen and hyperaemic.

Mesenteric lymph nodules considerably enlarged, not hyperaemic.

The *stomach* contains a considerable quantity of greenish black fluid, with minute, brownish black particles; blackish mucus is adherent to the mucosa, which shows intense hyperaemia of cardiac portion of anterior wall, and numerous petechiae. On the posterior wall are several small erosions, without any sign of haemorrhage.

Duodenum: slightly hyperaemic. Jejunum shows patches of hyperaemia; ileum pale, colon shows several hyperaemic patches.

Liver: 21 × 14 × 5 cms. Capsule normal; colour reddish grey, with yellow patches; substance yellowish grey, with patches of pronouncedly yellow colour, and a few patches of hyperaemia; consistence considerably diminished, and somewhat friable.

Pancreas: Slightly enlarged; the caudal portion shows intense hyperaemia, and some haemorrhage into the tissue; caput and corpus pale; the whole organ is of considerably diminished consistence.

Anatomical diagnoses: Hyperaemia meningum. Poly-lymphadenitis acuta. Metamorphosis adiposa myocardii, et hepatis. Pancreatitis acuta. Nephritis acuta incipiens duplex. Hypostasis pulmonum. Gastritis acuta ecchymotica. Hyperaemia jejuni and coli. Elytritis. Endometritis. Cystitis? Helminthiasis intestinalis. Tonsillitis acuta. Pharyngitis acuta.

Histological examination:—

Axillary lymph nodules: Slight diffuse hyperaemia, in some places more pronounced. One considerable haemorrhage and several smaller ones.

Myocard: Fragmentation, numerous small fat droplets in fibres.

Liver: Diffuse hyperaemia, one or a few large vacuoles (fat) in nearly every cell. Slight microcellular infiltration of periportal tissue and penetrating into the lobules.

Pancreas: Advanced necrobiotic changes.

Kidney: Numerous fairly small fat droplets in the epithelial cells near the basal membrane, mostly in the convoluted tubules, less in the straight tubules and only very few cells in the glomeruli.

The *spinal fluid* is clear, alkaline, gives fairly intense albumin reaction. The centrifugalized deposit consists chiefly of erythrocytes; also some lymphocytes and a few polymorphonuclear leucocytes are seen.

Microbiological examination:—

Peripheral blood: No parasites. *Heart blood*, nasgar tubes, long Gram-negative bacteria. *Spinal fluid*, nasgar tubes, short thick Gram-negative bacteria which change all the sugars tested. *Vomits:* The first portion examined is clear bile acid; second portion is reddish black, with suspended dark brown particles. It is intensely acid and contains erythrocytes (microscopical) and haemoglobin (Guajac test).

Group G contains four cases: Nos. 6, 26, 45 and 58. Case 6 was a typical case of infantile gastro-intestinal atrophia, and, when I saw the case, there was no suspicion whatever of 'vomiting sickness.' My attention was, however, drawn to this case, because it was said to have been diagnosed as 'vomiting sickness' about three weeks previously. I regard this as an example of mistaken diagnosis, which is apt to occur, especially in recovering cases. This case

I exclude absolutely from the discussion as far as 'vomiting sickness' is concerned, but it may be mentioned that even in this case meningeal hyperaemia was present, contrasting with the marked anaemia of all other organs. Case 26 was also one of general atrophia, but this time in an old man. In this case Gram-negative diplococci were cultivated from the spinal fluid, a result which there is no reason to consider otherwise than as an accidental post-mortem infection. Case 45 resembled the type of 'vomiting sickness,' but the presence of numerous malarial parasites affords sufficient explanation of the symptoms and of the anatomical lesions. This case serves to illustrate the difficulties of diagnosis and is therefore given in full. Malarial parasites were also found in Case 58, but in small numbers; the diagnosis of malaria as *causa efficiens* must therefore be regarded with some doubt, and a diagnosis of 'vomiting sickness' has to be considered. The patient in Case 58 was a sister to the one is Case 57 belonging to Group B, as organisms of the meningococcus type were found. The two sisters were taken ill and died at the same time, obviously from the same disease; it seems, therefore, that the essentially different results of the microbiological examinations must be disregarded and both cases classified as 'vomiting sickness.'

Case 6. J.R., 26 months, Porus. Ill for three weeks, with fever, vomiting and diarrhoea with green stools; no convulsions, no shrieks.

Post-mortem diagnoses: Atrophia ventriculi et intestinorum. Anaemia universalis. Hyperaemia piae matris cerebralis. Hypostasis pulmonum.

Case 26. R.L.D., about 60 years, black, from Belvedere, Portland, not seen before death, but reported as having been taken ill acutely with vomiting. Later it was found out that he had been weak for several months and had vomited for some days before death. The corpse was brought down to the hospital mortuary and a post-mortem examination performed, with Dr. George.

The autopsy on February 1, 1913, showed simple atrophy of all organs and multiple cysts in both kidneys. There were no signs of cerebral meningitis and no affection of the stomach. The spinal cord was not examined, but lumbar puncture gave no fluid, except a single drop, with which a nasgar tube was inoculated.

Histological examination:—

Liver: Atrophia pigmentosa.

Microbiological examination:—

Nasgar tubes, *spinal fluid:* Gram-negative diplococci in groups which change glucose, galactose, saccharose and maltose, but not lactose.

Case 45. M.K., 8 years, black, Spanish Town Road. Kingston Public Hospital, February 15, 1913.

Admitted at 5 p.m., suffering from vomiting and convulsions, which had started the same day, suddenly. Head retracted. Death at 7.20 p.m.

Post-mortem examination, at 12 noon, on February 16, about 17 hours after death :—Body fairly well nourished. No skin affection. Hyperaemia of lips and gums ; small ulcers on lips. No jaundice. Rigor present.

Cervical and axillary *lymph nodules* enlarged and hyperaemic, inguinal enlarged.

Lumbar puncture gives only one or two drops of serous, slightly blood-stained fluid. Spinal dura normal, pia hyperaemic ; cord normal in dorsal portion, the only part examined. Cerebral dura slightly hyperaemic, pia considerably hyperaemic, especially on convex surface ; a few drops of serous fluid at basis ; no fibrinous exudation, one or two c.c. of clear fluid in lateral ventricles ; brain substance normal, slightly pale.

Hypophysis small, 6 × 4 × 3 mms., normal in appearance.

Pleurae normal. Lungs show slight hyperaemia, most marked in postero-inferior parts. Bronchi hyperaemic, contain some mucous secretion ; trachea and larynx normal. Bronchial lymph nodules slightly enlarged and anthracotic.

Pericardium contains no fluid ; epicardium, endocardium and valves normal. Heart conus 6 × 7 × 2½ cms. ; left ventricle fairly well contracted. Myocardium normal. Pulmonary artery and aorta normal.

Thymus 6 × 4 × 1½ cms., normal, in appearance ; consistence slightly increased.

Thyroid body normal.

Peritoneum normal without fluid. Abdominal organs normally situated.

Spleen : 15 × 9 × 5 cms. Capsule slightly folded and thickened ; substance homogeneous dark purplish grey, without prominent follicles. Consistence considerably diminished.

Mesenteric lymph nodules slightly enlarged and hyperaemic.

Suprarenal capsules : Considerably hyperaemic, the left most pronouncedly so ; the left is also slightly enlarged and contains several small haemorrhages

Right *kidney :* 8 × 4½ × 2½ cms. Capsule easily detached ; surface smooth, with capillary hyperaemia ; some yellowish grey patches on surface ; cortex hyperaemic in superficial part, grey in columnar portion. Slight hyperaemia of pyramidal bases. Left kidney shows more marked hyperaemia.

Renal pelves, ureters and bladder slightly hyperaemic ; bladder contains about 30 c.c. of clear yellow urine.

Genital organs normal.

Liver : 25 × 16 × 5 cms. Capsule normal ; surface dark purple, with a few small yellowish patches ; structure and consistence normal. Bile ducts patent and normal ; gall bladder contains a considerable quantity of yellowish bile.

Pancreas normal in size ; slight hyperaemia ; considerably diminished consistence.

The *tongue's* entire surface is covered with a whitish fur. Left *tonsil* slightly enlarged, haemorrhagic infiltrated and contain some inspissated pus. Oesophagus normal. The *stomach* contains alimentary fluid and mucus. The mucosa shows a few hyperaemic patches, is otherwise normal. Duodenum shows hyperaemia and swelling of follicles. Jejunum shows patches of hyperaemia and slight follicular swelling in its upper part. The solitary follicles and Peyer's patches in the lower part of the ileum are swollen, but not hyperaemic. Appendix normal. Colon hyperaemic, with follicular swelling and dark, pigmented spots on follicles. No worms

Anatomical diagnoses : Hyperaemia meningum, pulmonum, intestinorum et capsulae suprarenalis. Splenitis acuta in chronica pigmentosa. Metamorphosis adiposa l.g. Hepatis et pancreatis. Gastro-enteritis follicularis. Poly-lymphadenitis acuta.

Histological examination :—

Axillary lymph nodules : Moderate hyperaemia.

Tonsils : Intense hyperaemia.

Liver : Slight fatty change.

Pancreas : Slight localized necrobiotic changes.

Kidney : Advanced necrobiotic changes of the epithelium of the convoluted tubules with karyolysis and granulation of protoplasm. No vacuoles, and glomeruli normal. Epithelia of straight tubules well preserved. Moderate irregularly distributed hyperaemia.

Microbiological examination :—

Spinal fluid : Nasgar tubes remain sterile.

Heart blood, smears : *Plasmodium vivax ;* abundant infection with young parasites.

Spleen : smears ; *Plasmodium vivax :* abundant pigmentation.

Heart blood : Nasgar tubes remain sterile. I haemoglobin-agar growth of Gram-negative cocco-bacilli.

Case 58. D.M.M., 4 years, black, seen, after death, with Dr. Peck, Spanish Town, on March 5, 1913.

Post-mortem examination :—

Lumbar puncture gives clear fluid, with admixture of blood. Dura normal. Pia slightly hyperaemic on lumbar portion of cord and convex surface of brain ; normal at basis. Lateral ventricles normal. Brain and cord normal.

Cervical, axillary and inguinal lymph nodules, enlarged and slightly hyperaemic, especially the axillary.

The *stomach* contains alimentary fluid ; slight hyperaemia of mucosa.

Duodenum shows considerable follicular swelling. Jejunum normal, except for a few hyperaemic patches. Ileum normal. Appendix and colon normal. One *Ascaris lumbricoides.*

Anatomical diagnoses : Hyperaemia piae matris l.g. Lymphadenitis acuta l.g. Hyperaemia pulmonum. Petechiae epicardii l.g. Hyperaemia ventriculi l.g. Duodenitis follicularis. Nephritis acuta l.g. Helminthiasis intestinalis.

Urine : Acid ; contains no albumin, sugar, bile pigment, or casts.

Histological examination :—

Axillary glands : Considerable hyperaemia, increase of stroma.

Spleen : Pigmentation.

Liver : Irregular hyperaemia ; marked fatty change.

Pancreas : Moderate fatty change.

Kidney : Moderate fatty change, some increase of stroma and some microcellular infiltration. Epithelia well preserved.

Duodenum : Brunner's glands prominent. Microcellular infiltration and irregular hyperaemia.

Microbiological examination :—

Spinal fluid, nasgar tubes : remain sterile. Heart blood, nasgar tubes remain sterile. Heart blood smears : *Plasmodium vivax*, sparse infection.

Group H includes five cases, Nos. 3, 21, 22, 28 and 53, in which I was unable to obtain sufficient data to attempt a diagnosis. These cases may be left entirely out of the discussion.

V. DESCRIPTION OF VOMITING SICKNESS

It is quite obvious, from the preceding report of cases, that not all of those described are of the same nature. An attempt to determine the nature of 'vomiting sickness' may be preceded by a re-grouping of the cases in order to establish a well-defined field for discussion. So far, it is necessary to include amongst the chief characteristics the negative one, that a diagnosis of vomiting sickness is inadmissible as long as a case can be diagnosed otherwise on clinical and pathological grounds. The microbiological examination does not necessarily decide this matter. Two or more diseases may be caused by the same pathogenic agent and nevertheless differ so essentially from each other that they are in all systems classified under different headings.

TABLE B

Vomiting Sickness			Possibly Vomiting Sickness				Yellow Fever	Malaria	Infantile or Senile Atrophy
Group A	Group B	Group C	Group D	Group E	Of Group F	Of Group G	Of Group F	Of Group G	Of Group G
16	12	12	5	3	4	1	1 (or 5)	1	2
40 cases			13 cases				1 (or 5) cases	1 case	2 cases

Furuncles, osteomyelitis, and generalised sepsis are often due to one and the same organism, but it would obviously be wrong to consider them as identical diseases. Similarly, 'vomiting sickness' might be caused by the same germ that is responsible for meningitis, for instance, and still with some right claim a name as independent disease. From this point of view we shall have to consider only the cases in Groups A, B and C. Those in the other groups either do not correspond to the type represented in A, B and C, or they are insufficiently observed, or they can be otherwise diagnosed.

It appears that an arrangement like that given in Table B would represent a natural classification of the provisionally established groups, leaving out of consideration the non-defined group H.

The following clinical and pathological description applies to the cases which I would regard as 'vomiting sickness.'

Definition.—'Vomiting sickness' is an acute infectious disease which chiefly attacks children, often with fatal issue in a very short time, and which has so far only been observed in blacks, coloured persons and Hindoos in Jamaica.

Etiology.—The question of a specific pathogenic germ has not yet been fully solved; it is discussed in the following section. Deficient hyperaemic conditions, malnutrition and exposure to cold have been mentioned by various observers as etiological factors of importance, but there is no evidence that any of these factors should be very essential. Personally, I feel inclined to eliminate malnutrition entirely from this list, as in my experience a considerable number of cases have occurred in well nourished, even fat, and absolutely healthy-looking individuals. The mode of transmission is unknown, and such suggestions as have been made in this respect are based more upon analogy than upon actual observation.

Distribution and epidemiology.—'Vomiting sickness' is, as far as the majority of the cases are concerned, a disease of people inhabiting poor huts more or less closely surrounded by bush. This, however, is by no means a rule without exception; several cases occurred in well-built and cleanly kept houses in villages, or situated on dry ground, apart from the bush.

Cases were observed, in this as in earlier outbreaks, in considerable numbers in some districts, and were altogether absent in others. This circumstance is, however, not one of very great importance. Districts are large in Jamaica, and it is no easy matter to trace the connection between ten and twelve cases scattered over an area of nearly a hundred square miles. As a matter of fact, a connection could rarely be demonstrated with certainty. When several cases occurred in one and the same house, a connection might of course be presumed to exist between them, but in each instance one would sooner think of a common source of infection than of transmission of the infection from one case to another, because the cases as a rule occurred either simultaneously or in very rapid succession. My material includes various examples of almost simultaneous occurrence of vomiting sickness in two or more members of one and the same family, living in the same house, as

in the following group of cases: Nos. 14, 15 and 16; 17 and child not observed; 35 and 36; 47, 48, and infant; 57 and 58. On the other hand, there are even more numerous instances of the isolated occurrence of the disease in one member of a family, although several other children were living in the same house.

Symptomatology.—The duration of the incubation period is unknown, but there is some reason to believe that it is short, considering the hyperacute character of the disease. Nothing definite has been recorded about the occurrence of prodromal symptoms.

The illness usually sets in suddenly, often in the night, or early morning, though this is by no means a constant feature; in fact, it is not uncommon that the symptoms are first observed in the evening and that death takes place during the night. Vomiting is very often the first symptom; it is usually watery or consisting of alimentary fluid, at any rate to begin with, but may later on become bilious or perhaps even haemorrhagic. Vomiting is not a constant, but an extremely common symptom; out of the forty cases in the Groups A, B and C, its occurrence is mentioned twenty-four times, and its absence six times; in the remaining ten cases no definite information was obtained. The next symptom, as regards frequency, is that of convulsions, which are sometimes described as clonic, but in other cases as tonic contractions, lasting for several minutes; an exact description of this symptom is at present hardly possible, as most cases were not medically observed. For the same reason, it is extremely difficult to discuss the occurrence and frequency of other nervous symptoms, but it can confidently be stated that coma is exceedingly common and that a sudden collapse apparently occurs in some cases. Rigidity of the neck muscles was not frequently observed, nor was rigidity of other muscles. Kernig's symptom was only recorded twice in the cases of the first three groups, but both with regard to this and the two foregoing symptoms it is important to remember that very few patients came under medical observation at all or sufficiently early to make a thorough examination possible.

Of other symptoms than those of a nervous character little is to be said. The course of the illness is so rapid that most organic lesions do not become recognisable clinically. Febrile temperatures

are seldom observed, but it seems probable that subnormal temperatures occur, though there is little evidence of this. The vomiting is, perhaps, in the majority of cases of cerebral origin, but just as often one would believe it to be due to gastric irritation. The severe renal affection which was frequently observed at the post-mortem examination could hardly be expected to produce any clinical symptoms; it may be specially mentioned that abundant urine was often found at the autopsy and that the amount of albumin was usually very small. It would appear that the urine found had been excreted before the development of the disease, or, at any rate, of the kidney affection. The rapidity with which the illness reaches a fatal termination shows, approximately, an inverse ratio to the age of the patients; infants die sometimes less than an hour after the appearance of the first symptoms, while cases in adults may last for one or two days.

Pathological anatomy.—The most striking anatomical lesions are: enlargement and hyperaemia of the lymphatic nodules, petechiae on the surface of the heart and in the gastric mucosa, necrobiosis of the pancreas, liver and kidneys, especially of the pancreas, and fatty change of the same and other parenchymatous organs. There is often a marked hyperaemia of the spinal and cerebral pia, but in other cases it was very moderate, and in others limited to the cerebral pia alone. The intensity of the lesions differed very much, especially that of the fatty changes, which was most marked in the cases of one or two days' duration in adults, whilst it was seen in its initial stage in cases of extremely rapid development.

General pathology.—The pathological physiology ought to explain the most striking feature of the disease, namely, the rapid course, the high mortality and the haemorrhagic degeneration and necrobiotic phenomena just described.

All these phenomena are readily explained as the results of an acute poisoning which may be due to the products of a bacterial or protozoal organism. If the vomiting is considered of cerebral origin, only the occurrence of cerebral symptoms has to be explained. All these symptoms, vomiting, convulsions and coma, may be due to an affection of the meninges, and this affection need not be a true meningitis; they may just as well be the expression of a state of 'meningismus.'

Mortality and prognosis.—According to some observers (Tillman and others, *Annual Report*) a very large number of cases of 'vomiting sickness' terminate in recovery. If this is true then many other observers, including myself, have only seen, or at any rate, have only diagnosed severe cases. This is possible, but for reasons stated above I consider it more likely that a large number of minor ailments, essentially different from 'vomiting sickness,' have been included in the statistics of the first-named observers. However, this controversy cannot be decided at present, and it may therefore be mentioned that the mortality has variously been found as low as about 2 per cent., and as high as about 100 per cent. In my own material, considering only the forty cases in Groups A, B and C, with thirty deaths, the mortality is 75 per cent. If a marked case of 'vomiting sickness' is seen, a very guarded prognosis should be given; recovery may take place, but the situation is always very serious.

Diagnosis.—Until the etiology of 'vomiting sickness' is more fully known, the diagnosis in vivo and post mortem must rest exclusively upon the symptoms described above.

Treatment.—No specific treatment can be recommended with any hope of success. Antimeningococcic serum might be used when 'meningococci' are found, but it is doubtful whether its use would be justified in other cases. Moreover, as Scott (1913, 2) has pointed out, its general use out in the country districts encounters many difficulties of a practical nature. An anti-serum might, perhaps, be prepared with the specific diplococcus, if this organism should finally be proved to be the cause of vomiting sickness. Vaccine treatment would hardly find any indication in this hyperacute disease. Hot baths and stimulating injections (ether, camphor, strychnine, etc.) may be recommended, but without much hope of success, especially as there will usually be very little time for the application of therapeutic measures.

Prophylaxis.—This question will be discussed in Section VII.

VI. THE NATURE OF VOMITING SICKNESS

At the onset of this discussion it is natural to ask, whether diseases identical to 'vomiting sickness' occur outside of Jamaica. Very little material is available which bears directly on this question.

In 1905 the Government of Jamaica made enquiries, in a circular letter to the authorities of other West Indian Islands, with regard to the possible occurrence of 'vomiting sickness' in other parts of the West Indies. Only two of the answers are quoted by Ker in the Annual Report (1906), the others being in the negative. The one positive answer is from Finlay, who mentions an outbreak of 25 similar cases with 5 deaths, amongst soldiers, in Cuba: he suggests that the disease is cerebro-spinal meningitis, no particulars are given in the quotation. The other positive answer deals with a disease (in Haiti) which has obviously no essential resemblance to 'vomiting sickness.'

Branch (1906), who has no personal experience with 'vomiting sickness,' does not admit it as a morbid entity, and is of opinion that similar cases occur elsewhere in the West Indies, carrying different names, and representing in reality well-known diseases, especially malaria and helminthiasis.

With regard to this opinion I must remark that it is difficult without personal observation to realize what 'vomiting sickness' means. Before seeing cases, I quite naturally had the same idea that it simply was something else; but the particular experience of seeing one fatal case following another in children who a few hours previously were playing out and who hardly developed any symptoms before they died, appeared to me almost unique, and left quite a different impression upon me.

Thus, until further evidence may be brought forward, we may assume that 'vomiting sickness' is a local disease of Jamaica.

Since the publication of Potter's and Scott's reports no further papers have appeared on the subject. It will now be justifiable to circumscribe the discussion, dealing, in the first place, with yellow fever and meningitis only.

With regard to yellow fever, some circumstantial evidence is in favour of this diagnosis. Jamaica was in earlier times one of the principal foci of the yellow fever infection, a veritable hot-bed for yellow fever. Even in this century, in 1904, a small outbreak occurred in Port Royal, which was apparently of spontaneous origin; this outbreak and the question whether there is any evidence that isolated, autochthonous cases occur, is discussed by Potter (1912).

Considering the question from an epidemiological point of view there is obviously good reason for investigating very carefully any disease which might prove an atypical form of yellow fever, and which might thus not only establish the fact of yellow fever endemicity in Jamaica, but also throw considerable light on yellow fever epidemiology in general. On the other hand, epidemiological evidence itself is almost sufficient to destroy this hypothesis. Yellow fever in native children in endemic areas is probably always a fairly benign disease; at any rate, there is no reason to suspect that it should ever acquire such a malignant, fulminant type as that of 'vomiting sickness.' The occurrence of these cases amongst the native population only, without ever attacking foreigners, is also very much against the theory of yellow fever. It might be argued that anti-yellow fever campaigns had made transmission impossible in the towns and that yellow fever had therefore become a disease of the bush instead of a town-disease, but even out in the country non-immune foreigners travel, apparently without ever suffering from attacks of yellow fever. Besides, cases of 'vomiting sickness' occurred, in my experience, in Kingston itself, and were common in the neighbouring district of Lower St. Andrew, which is much frequented by tourists, and in which many Europeans have their permanent residences. The seasonal incidence also is almost destructive to the hypothesis of yellow fever. Yellow fever occurs, without any doubt, at any time of the year in endemic areas, but it has never been known to show the exceedingly abnormal feature of dying out or becoming very rare during the hot and rainy seasons, when mosquitos, according to local information, are abundant, whilst, on the other hand, it prevails during the cool and dry months, during which, in my experience, mosquitos in general are rare, and *S. fasciata* very rare. Thus epidemiological evidence is rather against identifying 'vomiting sickness' with yellow fever; this should, therefore, only be done if the clinical and pathological evidence were absolutely overwhelming. But this is by no means so. The classical clinical symptoms of fatal yellow fever—as fever, black vomit, jaundice and anuria—are almost constantly absent. They may be absent, one or all, in cases of yellow fever, but it is hardly conceivable that they should all be absent in practically all cases in large outbreaks.

Fulminant yellow fever has been described, but not of the type which is met with in 'vomiting sickness.' The anatomical lesions in many cases of 'vomiting sickness' are certainly closely similar to those observed in yellow fever, but, then, I have previously (1911, 1) maintained that the anatomical diagnosis of yellow fever is even more difficult than the clinical one. No single pathological phenomenon is pathognomonic in yellow fever, but the combined picture of clinical symptoms and anatomical lesions is nearly always so. This combined picture, however, has not been met with in 'vomiting sickness.' With regard to the most important lesions it may be emphasized that haemorrhagic gastritis was common, but by no means constant in 'vomiting sickness,' and rarely as marked as in yellow fever; the affections of kidney and especially liver differed in showing more marked fatty changes and less marked necrobiotic phenomena than in yellow fever, especially when the comparatively slowly developing cases of 'vomiting sickness' are considered, as they naturally must be very particularly, as one would expect to find in such cases the most typical lesions. The Rocha-Lima type (see note on p. 438) of liver affection has not been marked in any case of 'vomiting sickness,' and, as a rule, not even a similar type has been observed.

The most striking feature of all is, perhaps, that these abnormal epidemiological, clinical and pathological characters, if the disease really were yellow fever, should have repeated themselves year after year in an absolutely typical manner. This certainly is a very strong argument which tends to show that we have to do with a specific and typical disease.

As regards parasitological evidence, little can at present be said. I had very rarely the opportunity of examining good blood smears taken whilst the patient was alive. In no case of typical 'vomiting sickness,' however, was the *Paraplasma flavigenum* found. I shall not here discuss the importance of the parasitological evidence in yellow fever, but must refer to my previous publications (especially 1911, 2 and 1912). The one case (Case 23) in which *P. flavigenum* was found, but which was not typical 'vomiting sickness,' has been discussed in the preceding section and will again be referred to in Section VIII.

The other disease with which 'vomiting sickness' has been, at

least partially, identified is cerebro-spinal meningitis. The evidence is that meningococci have been found by Scott (1912, 1 and 2, 1913, 1 and 2) in the cerebro-spinal fluid in a considerable number of cases of 'vomiting sickness.' Scott himself is, however, in his latest paper less positive with regard to the essential importance of his results, as far as 'vomiting sickness' is concerned, than he was to begin with. At any rate, there is more evidence in favour of the identity of 'vomiting sickness' with meningitis than with any other disease, and the question must be most seriously considered.

In the first place the bacteriological findings must be discussed. I shall not discuss the results published by Scott, but limit my remarks to my own investigations, in which I was, as stated above, greatly assisted by Dr. Scott himself. We fully realized—at least it soon became evident—the great difficulty in obtaining an absolutely reliable bacteriological diagnosis of meningococci, a difficulty which is, I believe, greater in Jamaica and probably in other tropical countries than elsewhere, as I shall presently explain. We therefore resolved to use the agglutination test, and not being in possession of a meningococcus strain from another locality used a strain obtained in one of Dr. Scott's first cases for immunization purposes. Unfortunately, however, the time at my disposal was too short; in fact, I gave the last of the commenced series of injections to several rabbits undergoing immunization, just before I left Jamaica. Thus I had to rely upon the morphological and cultural characters, staining reactions, and sugar reactions of the diplococci found. Of these characteristics, only the sugar reactions need a few preliminary remarks, as we, of course, only considered a diagnosis of meningococcus when colonies developed on ascites-agar, consisting of coffee grain-shaped diplococci, which did not retain the colour in the Gram-process. As regards the production of acid in sugar-media, there is no perfect agreement even between the most recent authors on the subject. Busse (1910) and Kutscher (1912) adopt Lingelsheim's view that meningcocci change glucose and maltose, but not other sugars, whilst Netter and Debré (1911), though in a general way confirming the same experiences, state that no very severe rules should be laid down. Scott (1913, 2) isolated in his first cases a diplococcus

which changed both glucose, maltose and galactose, leaving other sugars unchanged. These cocci were otherwise typical, and they were found in cases of anatomically well-characterized meningitis. For this reason we adopted these characters as a preliminary basis for classification, and according to this classification the distinction is made between the Groups A and B of my cases. The twelve strains isolated in the cases belonging to Group B showed the following reactions:—

No.	Glucose	Maltose	Galactose	Lactose	Saccharose	Mannite
1 ...	+	+	+	–	–	–
7 ...	+	+	–	–	–	–
8 ...	+	+	+	–	–	–
12 ...	+	+	+	–	–	–
20 ...	+	+	–	–	–	–
30 ...	+	+	+	–	–	–
33 ...	+	+	+	–	–	–
43 ...	+	+	–	–	–	–
44 ...	+	+	+	–	–	–
50 ...	+	+	+	–	–	–
55 ...	+	+	+	–	–	–
57 ...	+	+	+	–	–	–

Thus according to the classical rule, only the strains in Cases 7, 20 and 43 should represent typical meningococci. These three cases showed no more pronounced meningeal lesions than the others. In Group D, cases of meningitis, anatomically well characterized, the following reactions were obtained.

No.	Glucose	Maltose	Galactose	Lactose	Saccharose	Mannite
4 ...	+	+	–	...	–	–
19 ...	+	+	+	–	–	–
29 ...	+	+	+	–	–	–
31 ...	+	+	+	–	–	–
39 ...	+	+	+	–	–	–

In view of these results in the cases in Group D it appeared natural to uphold the distinction as previously stated.

No further unusual characters were observed during the examination of the cultures in Jamaica, but they became evident on re-examination of the cultures in Liverpool, where I continued several strains in Professor Beattie's laboratory. Already the survival of the cultures was remarkable. Some of them, which I brought home myself, survived twenty-seven days until sub-cultured. Other cultures, which Dr. Scott sent me, lasted for over two weeks. The following strains were examined: Cases 1 and 4 (A and B) and two of Dr. Scott's cultures, Stock I and Stock II. The following reactions were obtained:—

No.	Glucose	Galactose	Lactose	Saccharose	Mannite
1 (B.D.) ...	+	−	−	−	−
4 (V.C.) A ...	+	−	−	−	−
4 (V.C.) B ...	+	+	+	+	−
Stock I ...	+	−	−	−	−
Stock II ...	+	+	+	+	−

I now asked Dr. Ledingham, of the Lister Institute, for an immune serum for agglutination purposes. Unfortunately no rabbit immune-serum was available, but I received some of the Institute's anti-meningococcus horse serum, which Dr. Arkwright informed me showed marked agglutinating power, having been tested with numerous meningococcus strains. I likewise received normal horse serum from the Lister Institute. The results were as follows (macroscopical methods used):—

No.	Antimeningococcic serum						Normal horse serum
	1–20	1–40	1–80	1–160	1–320	1–640	1–20
1 (B.D.) ...	+++	+++	+	−	−	−	+++
4 (V.C.) A ...	+++	+++	−	−	−	−	++
4 (V.C.) B ...	+++	+++	+++	++	++	+	+++
Stock I ...	+++	++	+	−	−	−	+++

Only the strain 4 (V.C.) B gave a marked reaction; this result is remarkable, as this same strain gave quite atypical sugar reactions. It is, however, stated by several authors that there is a marked 'group reaction' amongst the meningococci and allied species. As the numbers of sub-cultures increased it was also observed that their aspects gradually changed; the growth was more abundant; and the colour more saturated whitish, characters which were particularly marked in Strain I (B.D.). The morphological characters remained the same, but the staining was somewhat irregular; it was found that on repeated occasions several of the strains showed a remarkable resistance to the decolorization after Claudius.

Dr. J. A. Arkwright also examined one of these cultures [4 (V.C.) B] and kindly informed me that he had observed considerable differences from the usual meningococcus strains, especially a considerably higher resistance. He found that the strain produced acid in glucose, maltose, galactose (slight) and cane sugar, but not in lactose.

Having learned that these strains possessed a considerable degree of variability, it appears advisable to revise Group A, as in many of the cases therein included diplococci were found, which were not considered in the discussion on meningococci on account of differences in sugar reaction or other characters. The following table shows these observations:—

No.	Glucose	Maltose	Galactose	Lactose	Saccharose	Mannite	Remarks
9	+	−	−	−	−	−	Diplococci forming chains
34	+	+	+	+	+	+	
37	+	+	+	±	−	−	
42	+	+	+	+	+	+	
46	−	+	−	−	−	−	Diplococci in chains

The strain in 37 showed but little difference from those previously described; in fact, sub-cultures gave no reaction in lactose media, which would perhaps justify including this case in Group B. The strains 9 and 46 might be considered typical

meningococci (according to Abel (1911) only glucose or maltose should be changed by meningococci), were it not for the peculiarity that the diplococci appeared in well marked chains. In this respect it resembles a strain which I found in a case of purulent meningitis observed in Yucatán (1910), and in which the formation of chains was a salient feature.

No pathogenic power was found by subcutaneous, intravenous, intraperitoneal and intracardial inoculations of living cultures of several strains into guinea-pigs or rabbits. These experiments need therefore not be detailed.

Thus, the bacteriological examination did not solve the question in an indisputable manner. The nature of the diplococci observed is not beyond question; but one thing seems evident; the distinction between Groups A and B, cases of 'vomiting sickness' type without and with meningococci, which was already at first but poorly marked, has been still more weakened by the closer examination of the cultures.

From the bacteriological data it is impossible to say whether the organisms isolated have any pathogenic importance or not. The organisms are not typical meningococci, but it is quite possible that they may cause pathological phenomena. I have, so far, come to conclusions similar to those of Arkwright (1909), who says about several organisms examined, 'They apparently have no relationship to the Meningococcus of Weichselbaum. They may, however, be the causal organism in some cases of meningitis, or may play a secondary part.'

The question, if possible to decide at the present time, must be decided on clinical, anatomical and epidemiological grounds. Clinically the typical cases of 'vomiting sickness' show considerable resemblance to fulminant cases of cerebro-spinal meningitis. Such cases have been described by many authors and are mentioned in recent text books. Thus, Osler (1912) says, 'Malignant form: This fulminant or apoplectic type is found with variable frequency in epidemics. It may occur sporadically. The onset is sudden, usually with violent chills, headache, somnolence, spasms in the muscles, great depression, moderate elevation of temperature, and feeble pulse, which may fall to fifty or sixty in the minute. Usually a purpuric rash develops. In a Philadelphia case, in 1888,

a young girl, apparently quite well, died within twenty hours of this form. There are cases on record in which death has occurred within a shorter time. Stille tells of a child of five years, in whom death occurred after an illness of ten hours; and refers to a case reported by Gordon, in which the entire duration of the illness was only five hours. Two of Vieusseux's cases died within twenty-four hours.'

'In malignant cases there may be no characteristic changes, the brain and spinal cord showing only extreme congestion, which was the lesion described by Vieusseux.'

Koplik (1907) mentions a malignant form of meningitis which is fatal within twelve, twenty-four or thirty-six hours. 'An adult or a child in apparent health complains of slight headache, there is nausea followed by vomiting, fever and unconsciousness in rapid succession. Death occurs in a short time, consciousness not being regained.'

A good description is also given by Ormerod (1905). This hyperacute form is particularly common in children, and is described in pediatric text books, for instance, Cautley (1910), Thiemich (1910) and Holt (1913). The latter says 'Cases of this kind are rarely seen except in an epidemic, and usually occur at its height. The onset is very abrupt, the course short and intense, and death may take place in from twelve to thirty-six hours. The following case illustrates this type: A little girl of ten years was well enough at 2 p.m. to carry a bundle of cloths a dozen city blocks. Returning home, she complained of intense headache, vomited frequently, and was so weak that she was obliged to go to bed. In a few hours she passed into deep coma, with very high fever and died at 11 p.m.' (p. 704.)

'There may be only a serous exudation and intense hyperaemia, which is doubtless much less marked after death than during life. The cerebro-spinal fluid is turbid and much increased in amount.'

The absence of definite inflammatory lesions of the meninges is recognized by the above authors and especially described by Busse (1910) and others. The whole question is particularly well dealt with by Netter and Debré (1911). Thus there is no doubt that such cases have been described, and so far there is no objection why the cases should not be diagnosed as hyperacute forms of meningitis.

With regard to the anatomical lesions of other organs than the meninges it may be mentioned that Busse (1910) lays stress upon the affection of the lymph-nodules, both peripheral and mesenteric. This corresponds to the conditions met with in vomiting sickness. On the other hand, such severe affections of liver, pancreas, and kidneys, as those found in my cases, have, as far as I know, not been described in cases of meningitis.

The typical case of 'vomiting sickness' corresponds fairly well to descriptions given of hyperacute cases of meningitis. It differs, however, as far as our present knowledge goes, in one important particular, namely, in the absence of fever. In the fulminant form of meningitis it is the rule that the temperature rises up rapidly, often to about 105°, or even more, whilst it is only mentioned as quite an exceptional thing that the temperature occasionally may remain normal until the fatal termination.

The epidemiological conditions, however, must also be considered. With regard to this question, practically all authors state that fulminant cases occur incidentally, isolated during epidemic outbreaks, and it is said by several, that they occur chiefly at the height of epidemics. The conditions in Jamaica are quite different. Here we have recurrent yearly outbreaks, and, perhaps, smouldering endemic prevalence.

It seems quite evident that no similar outbreaks of meningitis have ever been described. I therefore cannot accept at the present state of the investigations, the explanation, which at first sight seems the most probable, that 'vomiting sickness' is simply a form of meningitis. Experience has shown in several diseases that a germ which has for some time been regarded as a pathogenic one, has, on close investigation, been reduced to a secondary position as representing only a complicating infection. Further investigation is therefore absolutely imperative before the problem can be considered finally solved. It is quite possible that 'vomiting sickness' may be due to a blood-inhabiting, presumably protozoal, organism, and that a diplococcus infection of the meninges occurs as a frequent complication, as a rule not giving rise to any marked anatomical lesions, because of the rapidly following death, but in a few cases producing a typical fibrino-purulent meningitis. Another possibility is that the causal organism may be parasitic in

the intestine and produce exceedingly active toxins. Both explanations would correspond equally well to the clinical symptoms and both might also explain the anatomical lesions. The similarity of the latter to those observed in yellow fever makes one consider the existence of a blood parasite as very probable, but the marked affection of the lymph nodules in various parts of the body would, perhaps, be more in favour of a bacterial infection. In either case, whether the diplococci observed in these cases are the essential causal organisms or only of secondary importance, it seems, at any rate, very unlikely that they should be of no pathogenic importance at all. Whether these diplococci represent one or more species, it is impossible to say. At any rate they differ obviously from meningococci, gonococci, *Diplococcus crassus* and other known diplococci. The existence of more than one species is suggested by the very considerable differences in sugar reactions and agglutinating power. On the other hand, the great variability observed in some of the strains makes it possible to assume, until further investigation, the existence of only one new species. For this new species, which is very likely to be only a provisional one, I would suggest the name *Diplococcus jamaicensis*.

The chief characteristics of this species are that it is a diplococcus morphologically similar to gonococcus and meningococcus, as a rule Gram-negative, but occasionally decolourizing with some difficulty, showing fairly abundant growth on nasgar, the cultures being more resistant than meningococcus cultures, and changing at least glucose or maltose, but, as a rule, often one or several other sugars.

The mode of transmission of 'vomiting sickness' I have had no opportunity of investigating. As far as possible I examined the prevalence of mosquitos, ticks and other insects in houses which I visited with the object of seeing cases. Mosquitos were not commonly found, and when found were not abundant. Some were *Stegomyia fasciata*, some *Culex sp.*

I have not examined systematically cultures from the nasopharynx of contacts with 'vomiting sickness' cases, and I do not anticipate that very definite results can be obtained in this way until the etiology of 'vomiting sickness' is definitely settled. At present one would be in great difficulty with regard to a diagnosis between atypical strains of *Diplococcus jamaicensis* and

D. catarrhalis, and, further, one would not know how much importance should be attached to the positive results of the examination for *Diplococcus jamaicensis*.

Other theories, such as vomiting sickness being identical with malaria, with some form of helminthiasis, or with some kind of poisoning, find no support whatever in my observations.

VII. PROPHYLACTIC MEASURES

The absolute lack of knowledge about the mode of transmission of 'vomiting sickness' makes it impossible to lay down any rules with regard to prophylactic measures. On general hygienic principles it may be recommended that the poverty should be relieved and popular education, especially with regard to cleanliness, promoted. It is, of course, also important that anti-mosquito campaigns should be carried out, as well as campaigns against other insects, but to none of these measures can any specific importance be attached. It has been stated that no evidence points to insect transmission of the disease, and with regard to poor hygienic conditions it has also been stated previously that a considerable number of the individuals attacked by 'vomiting sickness' were absolutely healthy looking and well nourished; sometimes the houses and huts visited were also well kept and clean.

VIII. OTHER OBSERVATIONS IN JAMAICA

The one observation of a yellow fever case which has been mentioned in Section IV, would apparently tend to show that other cases might be of the same nature. There is, however, no evidence which makes it necessary to adopt this view. The cases occurred at the same time and in the same place as several cases of 'vomiting sickness,' but it showed a clinical course different from those cases, and the anatomical lesions were also different. There is, of course, no reason why the two diseases should not occur at the same time and in the same place, supposing that both exist in the Island. The problem is how to explain the occurrence of one

isolated case of yellow fever. This, however, is only what happens in many endemically but mildly infected places where the existence of yellow fever only becomes known when there is a considerable influx of foreigners. That outbreaks of yellow fever do not occur in Jamaica, though the disease may exist amongst the natives, is easily explained, I believe, by the relative freedom from mosquitos which the larger towns enjoy. The principal travelling public, the tourists, visit Jamaica during the winter months only, and at this time of the year I can safely assert that *Stegomyia fasciata* is not common in parts of Jamaica which I have visited, and which I believe in their natural conditions correspond fairly well with the various places which foreigners would visit.

I shall not mention here the laboratory observations on other subjects which were made during the 'vomiting sickness' investigation, but I want briefly to refer to a case which presented considerable interest, especially when considered in connection with my observations on Cutaneous Leishmaniasis in Yucatán (1912). This was a case of a fistulous and ulcerated tumefaction of the lower part of the femur, in which case I suspected infection with *Leishmania tropica*, but this parasite was never found, though repeatedly examined for. The particularly interesting point is that the exudation contained numerous intracellular diplococci, similar to those which were a constant feature in the cases of ear ulcer in Yucatán. Cultures were not obtained of this diplococci because of abundant mixed infection.

IX. OTHER PLACES VISITED

On my way to Jamaica and back I had the opportunity of visiting Trinidad, Barbados, the Panama Canal Zone, and Colombia. I visited the hospitals and saw interesting cases in Barbados and Trinadad and was fortunate in having a very good opportunity of seeing the sanitary arrangements on the Panama Canal Zone.

I also had the opportunity of discussing sanitary matters with Colonel Gorgas, Dr. Perry and other Officers of the Health Department, and was much impressed by the thoroughly well-established organization.

X. ACKNOWLEDGMENTS

The thanks of the Liverpool School of Tropical Medicine are due to those friends who generously provided the funds for the 'Vomiting Sickness' Expedition, to the Royal Mail Steam Packet Co. for granting free passages to and from Jamaica, as well as to the Leyland Line and Messrs. Elder & Fyffes for the free transport of goods.

I wish to express my gratitude to His Excellency Sir Sidney Olivier, Governor of Jamaica; His Excellency Mr. P. C. Cork, Acting Governor, and His Excellency Sir William Manning, Governor of Jamaica, who kindly arranged that all the facilities of the Health Department should be given to me for the furtherance of my investigations.

I am exceedingly indebted to the Hon. J. Erington Ker, Superintending Medical Officer, for the excellent arrangements which he made for reporting cases of vomiting sickness and for help in many other ways, as well as for great hospitality.

I am also greatly indebted to Dr. H. H. Scott, Government Bacteriologist, in whose laboratory I worked and who helped me in every possible way, as stated above.

I also wish to express my thanks to the following District Medical Officers:—Dr. Clarke, Dr. Cooke, Dr. Curphey, Dr. Earle, Dr. Edwards, Dr. George, Dr. Gifford, Dr. Joslen, Dr. Peck, Dr. G. W. Thomson, as well as to the Medical Officers of the Public Hospitals in Kingston, Dr. Castle, Dr. Ross, Dr. C. A. H. Thomson and Dr. Dryden, also to Dr. MacDonald (City Health Officer), Dr. Crosswell and Dr. Saunders.

TABLE I.—SUMMARY OF CASES OBSERVED IN JAMAICA, PRINCIPALLY

Case Number	Initials	Date	Age	Sex	Race	Locality	Emaciation	Vomiting	Black vomit	Convulsions	Kernig's symptom	Rigidity of neck-muscles	Rigidity of other muscles	Coma	Fever	Duration of illness (hours)	Clinical Character	Termination	Periph. blood	Heart blood	C. sp. fluid	Urine (alb.)	P.M. hours after death	Jaundice	Pia mater: Hyperæmia	Pia mater: Non-Inflammation	Brain	Spinal-cord	Peripheral Lymph Nodules: Enlarged	Mesenteric Lymph Nodules: Hyperæmia	Mesenteric Lymph Nodules: Enlarged	Spleen: Hyperæmia	Spleen: Enlarged	Spleen: Hyperæmia	Spleen: Hæmorrhage	Spleen: Inflammation	Liver: Hyperæmia	Liver: Hæmorrhage	Liver: Fat	Liver: Inflammation
1	B.D.	13/I	4	♀	Black	May Penn	–	–	...	+	...	...	+	...	...	3	V.S.	D	...	...	+	...	11	–	+	+	+	...	–	...	+	+	...	...	...	...	...	...	...
2	R.B.	13/I	4	♀	Coloured	May Penn	–	+	...	+	...	...	...	+	–	...	V.S.	R	...	...	+	...	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...
3	R.R.	13/I	2	♀	Coloured	May Penn	–	+	...	...	...	...	...	...	+	...	?	R	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
4	V.C.	14/I	2	♀	Black	Buff Bay	–	+	...	–	...	...	...	...	–	$\frac{1}{2}$	V.S.	D	...	...	+	...	25	...	+	–	+	...	–	...	+	–	...	...	...	...	...	...
5	A.A.J.	16/I	$1\frac{11}{12}$	♀	Black	Porus	+	–	...	–	...	...	...	...	–	2	V.S.	D	...	...	+	...	28	...	+	–	–	...	–	...	+	–	...	...	...	...	...	...
6	J.R.	18/I	$2\frac{5}{12}$	♂	Black	Porus	+	–	...	–	...	...	...	...	–	3 weeks	?	D	...	...	+	...	12	...	?	...	–	...	–	...	–	–	...	...	...	...	...	...
7	J.J.G.	18/I	8	♂	Black	May Penn	–	...	...	+	...	...	...	+	...	...	V.S.	D	...	...	+	...	26	...	+	–	–	...	–	...	–	–	...	...	...	...	...	...
8	H.S.	18/I	$3\frac{5}{12}$	♀	Black	Chapeltown	+	+	...	–	...	...	+	...	...	2	V.S.	D	...	–	+	...	16	–	+	–	–	–	–	...	–	+	–	–	...	...	...	...
9	A.S.	21/I	3	♀	Coloured	Kingston	–	+	...	–	–	–	–	–	–	48	V.S.	R	...	...	+	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
10	S.S.	21/I	4	♀	Coloured	Kingston	–	+	...	–	–	–	–	–	–	48	V.S.	R	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
11	A.W.	21/I	$\frac{12}{12}$	♀	Black	Kingston	...	+	...	–	–	–	–	+	+	48	V.S.	R	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
12	A.M.	21/I	3	♀	Black	St. Andrew	–	+	...	+	–	–	–	–	–	12	V.S.	D	...	...	+	...	16	...	+	–	–	–	–	...	+	+	...	–	–	...	...	...
13	T.S.	23/I	16	♀	Black	St. Andrew	–	+	...	...	–	–	–	–	–	48	V.S.	R	–	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
14	A.N.	28/I	12	♀	Coloured	St. Mary	+	+	...	–	–	–	–	+	+	72	V.S.	R	–	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
15	C.N.	28/I	9	♂	Coloured	St. Mary	+	+	–	–	...	...	...	+	–	20	V.S.	D	...	...	–	...	26	–	+	–	–	...	–	...	+	–	...	–	–	...	–	...
16	C.N.	28/I	$5\frac{6}{12}$	♂	Coloured	St. Mary	+	+	–	...	...	...	...	+	–	20	V.S.	D	...	...	–	...	26	–	+	–	–	...	–	...	+	–	...	–	–	...	–	...
17	M.H.	29/I	27	♀	Coloured	St. Andrew	–	+	...	–	–	–	–	+	+	60	V.S.	D	–	...	–	...	$1\frac{1}{2}$	–	+	+	–	–	–	...	–	–	–	–	–	–	–	–
18	A.B.	29/I	$\frac{12}{12}$	♀	Black	St. Andrew	...	–	...	+	–	–	–	–	–	?	?	D	...	...	–	...	...	–	+	–	...	...	...	...	...	...	...	...	...	...	...	...
19	A.H.	18/I	4	♂	Black	Kingston	–	+	...	+	+	+	+	–	–	...	V.S.	D	...	...	+	...	...	...	+	+	...	...	...	...	+	...	...	...	...	...	...	...
20	T.J.	31/I	12	♀	Coolie	St. Mary	...	...	...	...	...	...	...	...	...	10	V.S.	D	...	–	+	...	12	...	+	–	...	...	...	+	...	...	...	+	–	...	...	...
21	E.W.	31/I	60	♀	Black	Portland	...	+	...	–	...	...	...	...	–	...	V.S.	R ?	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
22	I.B.	1/II	42	♀	Black	Portland	–	+	...	–	–	+	...	+	–	48	V.S.	D	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
23	C.C.	31/I	20	♂	Chinaman	Portland	–	+	+	...	...	–	...	+	?	5 days	...	D	P.	...	...	+	$2\frac{1}{2}$	...	+	–	–	...	–	...	–	–	...	–	–	...	–	...
24	M.E.	1/II	Adult	♀	Black	Portland	...	+	...	–	–	–	–	–	–	3 days	V.S.	R ?	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
25	R.M.	31/I	12	♂	Black	Portland	...	+	...	...	–	–	...	...	...	24	...	R	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
26	R.L.D.	1/II	60	♂	Black	Portland	+	+	...	...	...	...	...	...	...	?	...	D	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
27	J.M.	1/II	...	...	Black	Portland	...	...	...	...	...	...	...	...	...	...	...	R	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
28 ?	...	...	...	...	...	Portland	...	...	...	...	...	...	...	...	...	...	V.S.	R	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
29	B.W.	...	12	♂	Black	Kingston	...	+	...	...	–	–	–	...	...	...	V.S.	D	M.	...	+	...	...	...	+	+	...	...	...	...	...	...	...	...	...	...	...	...
30	D.B.	31/I	$\frac{10}{12}$	♀	Black	Kingston	...	+	...	+	+	+	+	–	–	...	•	D	...	...	+	...	...	...	+	–	...	...	...	...	...	...	...	...	...	...	...	...
31	H.M.	3/II	$1\frac{1}{12}$	♀	Black	Kingston	...	+	...	+	?	+	+	+	+	...	•	D	...	...	+	...	...	...	–	+	...	...	...	...	...	...	...	...	...	...	...	...
32	G.B.	5/II	6	♀	Black	Fourpaths	+	+	...	...	...	...	+	...	...	...	V.S.	D	...	–	+	...	26	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
33	E.B.	5/II	2	♂	Black	Fourpaths	+	–	...	–	...	...	...	...	...	...	V.S.	D	...	...	+	...	25	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
34	R.S.	6/II	4	♂	Coolie	St. Catherine	–	+	...	+	–	–	–	–	+	...	V.S.	D	...	–	+	...	4	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
35	E.M.	6/II	11	♀	Black	Tollgate	–	+	?	+	...	+	+	...	...	...	V.S.	D	...	...	...	...	28	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
36	P.B.	6/II	11	♀	Black	Tollgate	+	+	–	–	...	+	...	...	...	...	V.S.	D	...	...	...	...	23	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
37	G.B.	7/II	$1\frac{4}{12}$	♂	Black	Fourpaths	...	...	...	...	...	+	...	...	...	...	•	D	...	...	...	...	36	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
38	D.L.	8/II	8	♂	Black	Kingston	...	?	?	...	...	...	...	+	...	...	...	D	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
39	U.M.	11/II	8	♂	Black	Fourpaths	+	+	...	...	...	...	...	...	...	...	V.S.	D	...	...	...	+	19	...	+	+	–	...	–	...	+	–	...	–	–	...	–	...
40	A.M.Y.	12/II	$2\frac{1}{2}$	♀	Black	St. Andrew	–	–	...	...	...	...	+	–	...	...	V.S.	D	...	•	+	...	24	...	+	+	–	...	–	...	+	–	...	–	–	...	–	...
41	M.A.B.	14/II	3	♂	Black	St. Andrew	...	+	...	+	...	...	...	...	...	...	V.S.	D	...	–	–	+	24	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
42	C.W.	14/II	$2\frac{1}{2}$	♀	Black	St. Andrew	...	+	...	+	...	...	...	+	...	...	V.S.	D	...	•	+	–	7	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
43	C.B.	14/II	2	♀	Black	St. Andrew	...	+	...	+	...	...	...	...	...	...	V.S.	D	...	•	+	...	15	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
44	E.P.	15/II	2	♂	Black	St. Andrew	...	+	...	+	...	...	...	...	...	...	V.S.	D	...	•	+	...	15	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
45	M.K.	16/II	8	♀	Black	St. Andrew	...	+	...	+	...	...	...	...	...	...	V.S.	D	M.	•	–	...	17	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
46	E.B.	17/II	$2\frac{1}{2}$	♀	Black	St. Andrew	...	...	...	+	+	+	+	+	...	...	V.S.	D	–	•	+	...	19	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
47	A.W.	18/II	20	♀	Black	St. Andrew	...	+	–	–	–	–	+	+	...	...	V.S.	D	–	•	+	...	13	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
48	L.W.	18/II	12	♂	Black	St. Andrew	...	+	...	...	–	–	–	...	...	...	V.S.	R	–	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
49	M.A.W.	19/II	3	♀	Black	May Penn	...	+	...	+	...	...	...	+	...	...	V.S.	D	–	...	...	...	15	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
50	J.M.	21/II	3	♀	Black	St. Andrew	...	...	...	+	–	–	–	+	...	...	V.S.	D	–	...	...	...	17	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
51	E.E.E.	23/II	20	♀	Black	St. Andrew	...	...	–	+	–	+	...	+	+	...	V.S.	D	–	...	–	...	19	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
52	J.E.	24/II	6	♀	Black	St. Andrew	...	...	...	+	...	...	...	+	...	...	V.S.	D	...	...	...	...	33	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
53	...	23/II	...	♂	Black	St. Andrew	...	+	...	...	...	...	...	+	...	...	?	R	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
54	D.A.	25/II	7	♀	Coloured	St. Andrew	...	+	+	+	...	...	...	+	...	...	V.S.	D	...	...	...	...	19	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
55	A.T.	2/III	6	♂	Black	St. Andrew	...	+	–	–	+	+	+	...	...	...	V.S.	D	...	...	–	...	14	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
56	R.W.	3/III	...	♂	Black	Kingston	...	...	...	...	...	...	...	...	...	...	V.S.	D	–	...	...	...	...	...	+	–	–	...	–	...	+	–	...	–	–	...	–	...
57	B.M.	5/III	5	♀	Black	Spanish Town	...	...	...	...	...	...	...	...	...	...	V.S.	D	...	...	...	...	...	...	+	–	–	+	–	...	+	–	...	–	–	...	–	...
58	D.M.M.	6/III	4	♀	Black	Spanish Town	...	...	...	...	...	...	...	...	...	...	V.S.	D	M.	...	–	...	21	...	+	–	–	+	–	...	+	–	...	–	–	...	–	...
59	C.W.	6/III	3	♀	Black	St. Andrew	...	...	...	...	...	...	...	...	...	...	V.S.	D	...	...	–	...	8	...	+	–	–	+	–	...	+	–	...	–	–	...	–	...
60	I.R.	7/III	$4\frac{1}{2}$	♀	Black	St. Andrew	...	+	...	+	...	...	...	+	...	...	V.S.	D	...	...	–	...	9	...	+	–	–	+	–	...	+	–	...	–	–	...	–	...
61	C.W.	8/III	12	♂	Black	St. Andrew	...	–	...	+	...	...	...	+	...	...	V.S.	D	...	...	–	...	31	...	+	–	–	+	–	...	+	–	...	–	–	...	–	...
62	R.A.L.	9/III	9	♀	Black	St. Andrew	...	...	...	...	...	...	...	...	...	...	V.S.	D	...	...	–	...	...	...	+	–	–	+	–	...	+	–	...	–	–	...	–	...
	Column No.		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)	(21)	(22)	(23)	(24)	(25)	(26)	(27)	(28)	(29)	(30)	(31)	(32)	(33)	(34)	(35)	(36)	(37)	(38)

V.S. = Vomiting Sickness D = Death R = Recovery M. = Malaria P. = Paraph

...BSERVED IN JAMAICA, PRINCIPALLY VOMITING SICKNESS

		Mesenteric Lymph Nodules		Spleen			Liver					Pancreas					Kidneys							Stomach			Duodenum			Intestines								Lungs			
		Hyperaemia	Enlarged	Hyperaemia	Haemorrhage	Inflammation	Hyperaemia	Haemorrhage	Fat	Inflammation	Necrosis	Hyperaemia	Haemorrhage	Fat	Necrosis	Inflammation	Hyperaemia	Haemorrhage	Fat	Necrosis	Inflammation	Adrenals	Tonsils	Hyperaemia	Petechiae	Black content	Hyperaemia	Petechiae	Inflammation	Hyperaemia	Petechiae	Inflammation	Ascaris	Tricocephalus	Ankylostoma	Pericard petechiae	Myocard fatty change	Hyperaemia	Ecchymoses	Infarcts	Oedema
		–					+		+	+	–	...	–		–	.	.	+	–	–	+	–	–	+	+	–	–	–	–	+	–	+	7	few	–	.	–	–	–	.	–
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(29)	(30)	(31)	(32)	(33)	(34)	(35)	(36)	(37)	(38)	(39)	(40)	(41)	(42)	(43)	(44)	(45)	(46)	(47)	(48)	(49)	(50)	(51)	(52)	(53)	(54)	(55)	(56)	(57)	(58)	(59)	(60)	(61)	(62)	(63)	(64)	(65)	(66)	(67)	(68)	(69)	(70)

M. = Malaria

P. = Paraplasma flavigenum

• = vide Text

§ Tuberculosis of lungs and other organs

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EXPLANATION OF PLATES

PLATE XXIX

Figs. 1-2. Two huts in which cases of vomiting sickness occurred. These figures show the very primitive structure and poor appearance of many of the native huts and their situation in the bush.

Plate XXX

All figures are from safranin-stained sections of specimens fixed in Fleming's strong osmic acid solution, and all are drawn by means of Abbe's drawing apparatus, the optic system being Zeiss's apoch. 4 mm. and compensating ocular 4. × 360. All figures on this and the following plate have been drawn by Mrs. Margrethe Seidelin.

Fig. 1. Stomach. Marked fatty change of epithelial cells.

Fig. 2. Pancreas. Marked fatty metamorphosis; small and medium-sized fat droplets are present in practically all the epithelial cells of the alveoli. A Langerhan's islet is shown in the figure, but it is not very sharply defined; its cells have undergone fatty change, but less marked than in the case of the alveoli. The epithelia of the excretory ducts are in particular severely affected.

Fig. 3. Myocardium. Moderate fatty metamorphosis.

Fig. 4. Liver. Intense fatty metamorphosis.

Plate XXXI

Technique as indicated for Plate XXX (× 360).

Fig. 1. Suprarenal capsule (Case 55) cortex. A considerable amount of fat is present in nearly all epithelial cells.

Fig. 2. Kidney (Case 62). Moderate fatty change, both convoluted tubules and glomeruli cells being affected, the latter less than the former, but in the case of both only minute fat droplets are observed in the cells.

Fig. 3. Kidney (Case 47). Intense fatty metamorphosis, the epithelia of the convoluted tubules contains very large fat drops; in the lining epithelia of Bowman's capsules and the glomeruli cells fatty change is also observed, but much less marked.

Fig. 4. Kidney (Case 47). Intense fatty metamorphosis of the epithelia of the ascending limbs of Henle's loops, whilst in the descending limbs the change is very little marked, only some of the epithelial cells showing minute fat droplets.

Plate XXXII

Microphotographs

Fig. 1. Mucosa of stomach (Case 55), showing hyperaemia, diffuse lymphoid infiltration, and superficial necrosis and haemorrhages. To the right, part of a lymphoid follicle is seen. × 95.

Fig. 2. Mucosa of duodenum (Case 34), showing hyperplasia of Brunner's glands and diffuse microcellular infiltration; also superficial necrosis. Remnants of the muscularis mucosae are seen, but this coat has been entirely broken up by the proliferated glands. × 95.

Fig. 3. Liver (Case 55), showing intense diffuse fatty metamorphosis and moderate necrobiotic changes, likewise of a diffuse character (× 370). Other parts of the same liver showed intense capillary hyperaemia.

Fig. 4. Pancreas (Case 43), showing marked necrobiotic changes. × 370.

FIG. 1

FIG. 2

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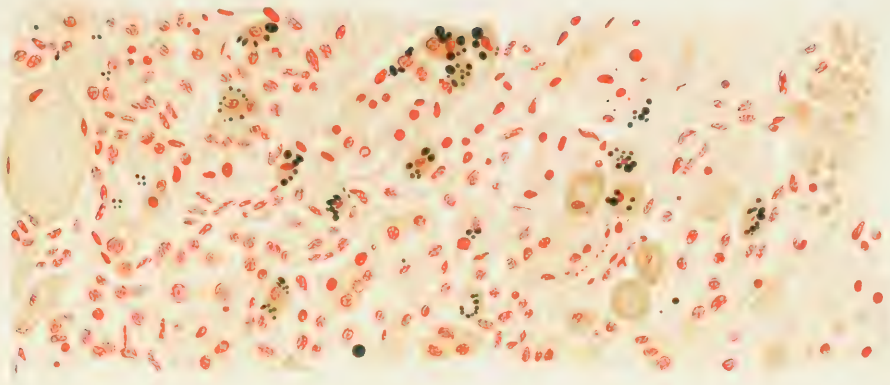
1



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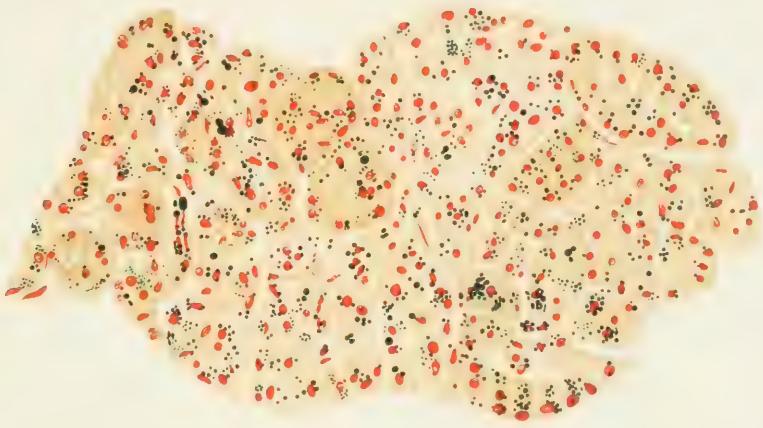

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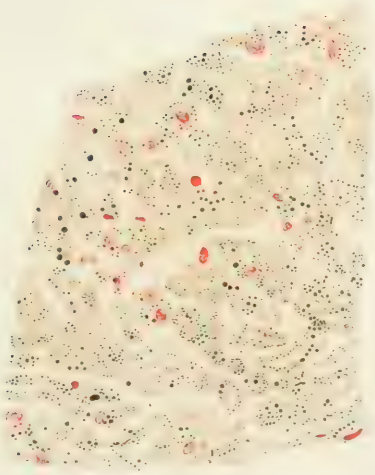

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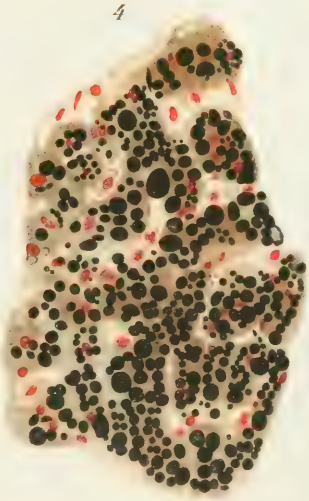

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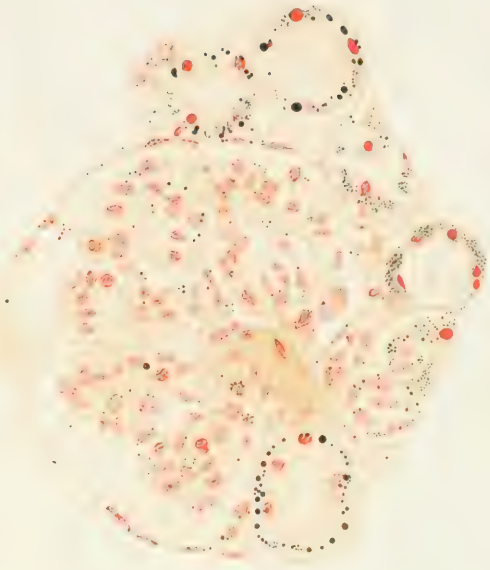

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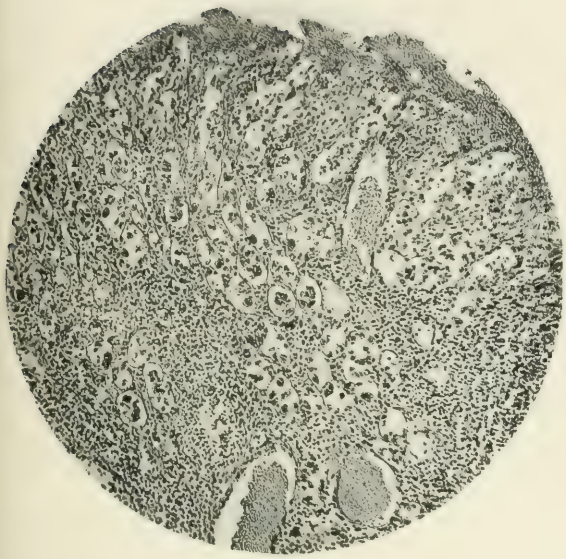
Margrethe Seidelin pinx.

PLATE XXXII



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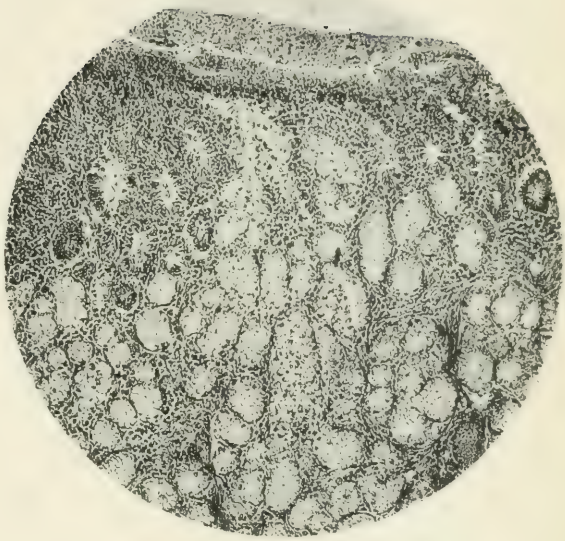

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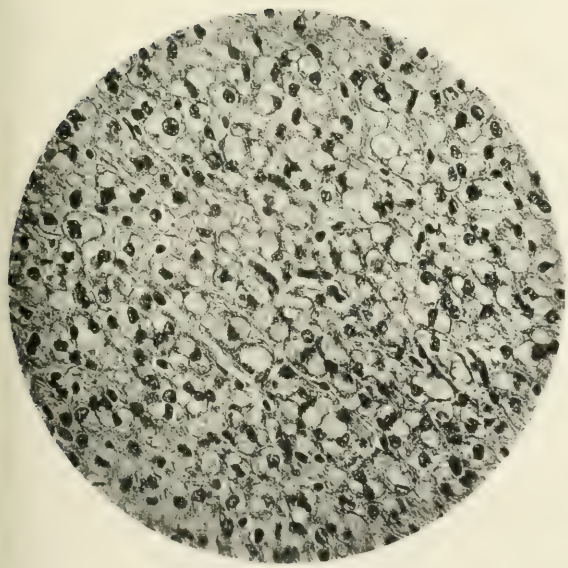

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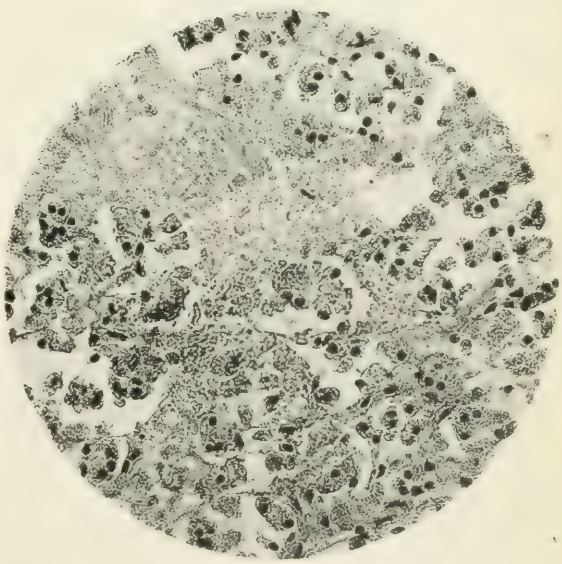

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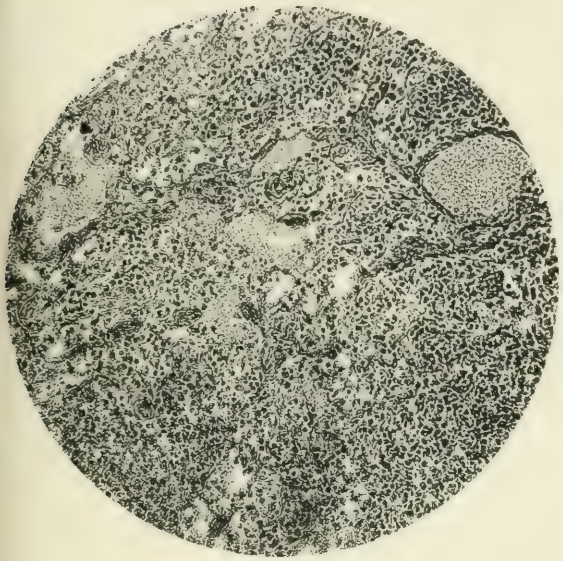

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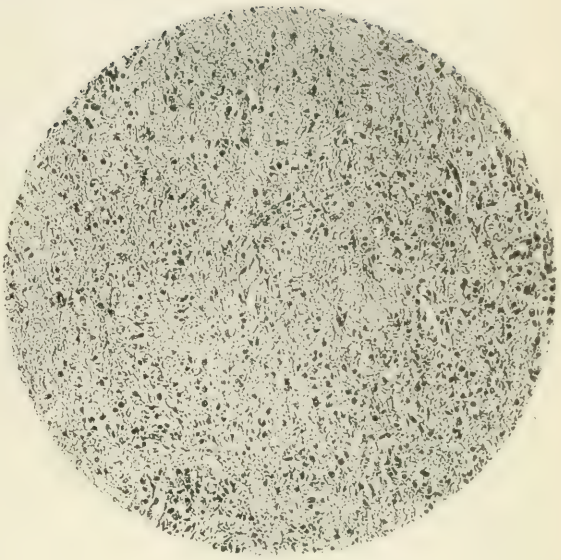

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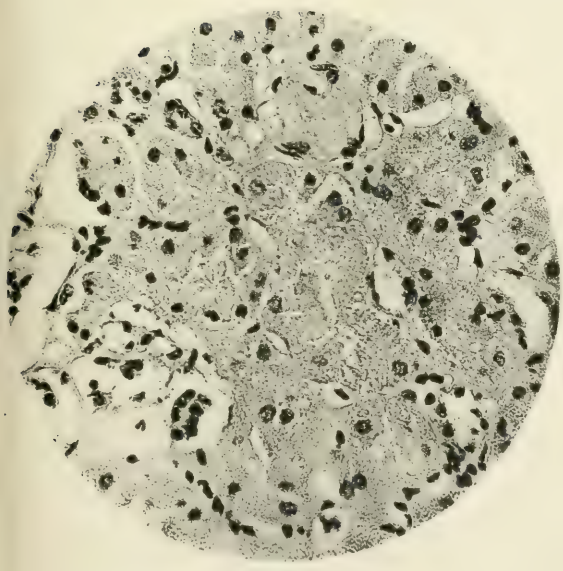

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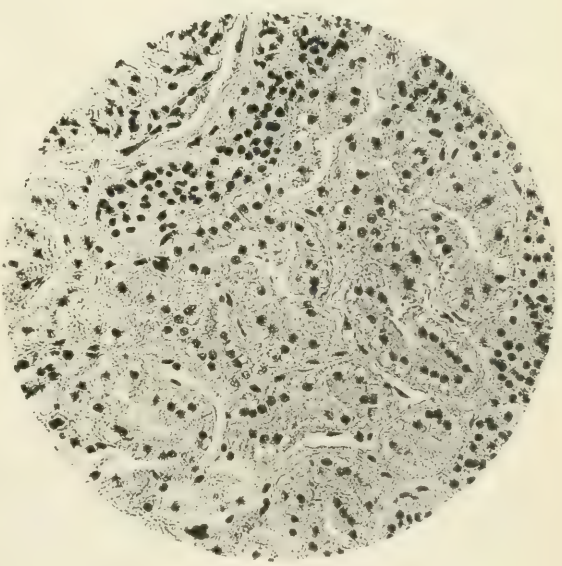

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